THESIS

FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY (General Surgery)

SESSION 2020-2023

"PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES"

DR. ABHINAV MONE

SANTOSH DEEMED TO BE UNIVERSITY
NO. 1, SANTOSH NAGAR,
GHAZIABAD-201009, NCR DELHI, INDIA

DEPARTMENT OF GENERAL SURGERY
SANTOSH MEDICAL COLLEGE & HOSPITAL
GHAZIABAD, NCR DELHI

THESIS

FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY (General Surgery)

SESSION 2020-2023

"PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES"

DR. ABHINAV MONE

SANTOSH DEEMED TO BE UNIVERSITY
NO. 1, SANTOSH NAGAR,
GHAZIABAD-201009, NCR DELHI, INDIA

DEPARTMENT OF GENERAL SURGERY
SANTOSH MEDICAL COLLEGE & HOSPITAL
GHAZIABAD, NCR DELHI

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

COPY RIGHT CERTIFICATE-DECLARATION BY THE CANDIDATE

I hereby declare that Santosh Deemed to be University, Ghaziabad, (U.P.) shall have the right to preserve, use and disseminate this thesis titled **"Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries"** in print or electronic format, for academic or research purpose.

Signature of Candidate:

Date:

Place:

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

SANTOSH UNIVERSITY

DECLARATION BY THE CANDIDATE

I hereby declare that this thesis titled "**Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries**" is a bonafide genuine research work carried out by me under the esteemed guidance of my Guide **Dr. Tripta S. Bhagat**, Professor in the Department of General Surgery, Santosh Medical College & Hospital, Ghaziabad, U.P.

Signature:

Date:

Place:

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

CERTIFICATE BY THE GUIDE

This is to certify that the thesis titled "**Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries**" is a bonafide genuine research work carried out by **Dr. Abhinav Mone** in partial fulfilment of the requirement for the degree of M.S. General Surgery under my Supervision and guidance in department of surgery, Santosh Medical College & Hospital, Ghaziabad, U.P.

GUIDE: **Prof. (Dr.) Tripta S. Bhagat**

M.B.B.S., M.S.

Professor

Department of General Surgery

Santosh Medical College & Hospital, Ghaziabad- 201001

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

CERTIFICATE BY THE CO-GUIDE

This is to certify that the thesis titled "**Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries**" is a bonafide genuine research work carried out by **Dr. Abhinav Mone** in partial fulfilment of the requirement for the degree of M.S. General Surgery under my Supervision and guidance in department of surgery, Santosh Medical College & Hospital, Ghaziabad, U.P.

CO- GUIDE: **Prof. (Dr.) Shalabh Gupta**

M.B.B.S., M.S.

Professor

Department of General Surgery

Santosh Medical College & Hospital, Ghaziabad- 201001

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

CERTIFICATE BY THE CO-GUIDE

This is to certify that the thesis titled "**Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries**" is a bonafide genuine research work carried out by **Dr. Abhinav Mone** in partial fulfillment of the requirement for the degree of M.S. General Surgery under my Supervision and guidance in department of surgery, Santosh Medical College & Hospital, Ghaziabad, U.P.

CO- GUIDE: **Prof. (Dr.) Neelima Agarwal**

M.B.B.S., M.D. OBS & GYN

Professor

Department of OBS & GYN

Santosh Medical College & Hospital, Ghaziabad- 201001

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

CERTIFICATE BY THE HEAD OF DEPARTMENT

This is to certify that the thesis titled "**Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries**" being submitted by Dr. Abhinav Mone in partial fulfillment of the requirement for the degree of M.S. General Surgery is based on the bonafide work carried out by him in Department of surgery, Santosh Medical College & Hospital, Ghaziabad, U.P.

Prof. (Dr.) Sameer Q. Naqash

M.B.B.S., M.S.

Professor & Head

Department of General Surgery

Santosh Medical College & Hospital, Ghaziabad- 201001 (UP)

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

ENDORSEMENT BY THE DEAN OF THE COLLEGE

This is to certify that the thesis titled **"Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries"** is a bonafide genuine research work carried out by **Dr. Abhinav Mone** himself under the esteemed guidance of **Dr. Tripta S. Bhagat**, Professor, General Surgery, Santosh Medical College & Hospital Ghaziabad.

Dr. Alka Agarwal

MBBS, MD

Dean

Faculty of Medical Sciences

Santosh Medical College & Hospital,

Santosh Deemed to be University.

Ghaziabad-201001 (UP)

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

ANTI-PLAGIARISM DECLARATION/UNDERTAKING

I, **Dr. Abhinav Mone**, hereby declare that the present thesis titled **"Pre-operative serum albumin as a predictor of post-operative morbidity and mortality in major general surgeries"** under the guidance of **Dr. Tripta S. Bhagat** at Santosh Deemed to be University is a bonafide/original research work. I also hereby declare that there is less than 10% similarity and the present research work has not been submitted for the award of any degree/diploma of the same institution where the work was carried out or at any other institution.

Signature of the Guide **Signature of the Student**

I have verified the contents of the thesis and it has less than 10% similarity.

Dr. Jyoti Batra

Dean Research & Professor of Biochemistry

Santosh Medical College & Hospital

Santosh Deemed to be University

Ghaziabad – 201001(U.P.)

ACKNOWLEDGEMENT

"Success is determined not by whether or not you face obstacles, but by your reaction to them. And if you look at these obstacles as a containing fence, they become your excuse for failure. If you look at them as a hurdle, each one strengthens you for the next."

- Benjamin Solomon Carson Sr.

Having completed my under-graduation around 7 years ago, I have had roller coaster ride of my life before I could attain my place in this institution for the degree like many other students, of my dreams. All my family, relatives, friends, seniors, employers and all other well-wishers, whom I have been blessed to have in large numbers, tried to convince me, either out of love or anger or frustration or reasoning, to either give up on my dream subject or compromise on it and look for alternatives. But the pleasure of this success is not only soothing over all my past failures but also immensely overwhelming for all my loved ones including me.

The road to success is never easy but once achieved all roads to the goal seems like a bed of flowers giving you fragrance of knowledge and divinity of experience.

With a heartful modesty, it is a genuine pleasure to express my deep sense of thanks and gratitude to my mentor, teacher and Guide ***Dr. Tripta S Bhagat****, Vice-chancellor and Professor Department of General Surgery. It has been my delightful pleasure to walk under the shadow of such an eminent advisor and mentor for my study. I am grateful for her unvarying support, timely counsel, meticulous inspection, scientific approach and scholarly advice and guidance throughout my post-graduation which has helped me accomplish this research. She has always been approachable, understanding, calm and reasonable to our problems and with nippy and witty solutions for the same.*

I would also thank from bottom of my heart to ***Dr. Shalabh Gupta****, Dean Academics & Professor of General Surgery for becoming my Co-guide and supporting me every time. His presence and guidance made this research a wonderful journey.*

I owe a deep sense of gratitude to ***Dr. Neelima Agrawal****, Professor of Obstetrics & Gynaecology who is also my Co-guide in this thesis. I would like to thank her for all the opportunities, guidance, insightful comments and encouragement. Her support and knowledge have helped me achieve this timely thesis. This same amount of support and guidance was also provided by all the seniors, my colleagues and juniors of the surgery department who helped me through my thesis work and for which I shall always be in debt.*

I would also like to extend my thanks and appreciation for all my Consultants ***Dr. Sameer Q Naqash, Dr. Gyanendara S. Mittal, Dr Aparajeeta, Dr. Amit Malik, Dr. Prakhar Garg, Dr. Atul Sharma, Dr. Sandeep Moolchandani, Dr. Siddharth, Dr. Rachit Goel, Dr. Rajiv Verma, Dr. Anupam Sharma, Dr Shishir Kumar, Dr. Rahul Gautam, Dr Sachin Chaudhary, Dr Naveen Solanki, Dr. Anu Vibhu, Dr. Kartik Kaistha and Dr. Saikh Kasif Sahajada*** *for their support and valuable comment. They also have been role models in many ways commendable with learning in every step of our journey in this institution thus leaving a lasting impression on our mind n souls.*

I would like to thank all my Seniors ***Dr. Abhishek, Dr. Anshul, Dr. Alok, Dr. Jebin, Dr. Sagar, Dr. Angelin and Dr. Priyanka Jadaun****; this thesis could not have been completed without their help and support.*

*My heartfelt thanks to my Colleagues **Dr. Janani, Dr. Sayandeep, Dr. Nithin, Dr. Ruksana, Dr. Riya, Dr. Nida, Dr. Tanuj, Dr. Priyanka and Dr. Madan** who over these years have become like an extended family in a place far from home. With their help and support this journey has become memorable and easy sail thorough the tidy waves.*

*I would like to thank all my juniors (**Dr. Adhar, Dr. Mansi, Dr. Deep, Dr. Sudhakar, Dr. Shilpa, Dr. Soha, Dr. Shreyansh, Dr. Rohan, Dr. Shrishti and Dr. Austin**) this thesis could not have been completed without their help and support.*

I shall always cherish my association with Department of General Surgery, Santosh Medical College & Hospital, Ghaziabad where I had this opportunity of carrying out the present study and where I have learned and benefited immensely to become a good Surgeon.

I would also avail this opportunity to thank all my friends here and back home who have been a constant shore for all my ups and downs in this memorable voyage. Success has no meaning if you are unable to share it with our loved ones. Their happiness in your triumph adds stars to the moony nights of your celebration.

*I owe a great debt to mother **Mrs. Jyoti Mone** and my father **Mr. Sunil Mone**. I feel myself lucky, for having such caring understanding parents whose love and guidance throughout my life has shown me the right path, motivate me to work hard and achieve the goals when I felt disheartened or lost and to cherish every big and small achievements in this exceptionally enlightening phase of my small life. Their love and blessings always helped me and kept my dreams alive.*

*I owe a big thanks to my wife **Radhika**, and daughter **Amyra** for supporting all the time in preparing my thesis. I would also extend my thanks to **Mr. Sanjay Deopujari** and **Mrs. Neelima Deopuja**ri my in-laws for their support and love during these academic years.*

No study can be completed without the co-operation of the patients involved and hence I express my sincere thanks and respect to all my patients from whom I have learned so much during this research. Their righteous feedbacks have been the true essence of my research.

Above all, I remember our divine GODs for giving me this opportunity, and blessing me with such empowering teachers, institution, intellect and skill in accomplishing this work as success.

Dr. Abhinav Mone

(Batch 2020-2023)

CONTENTS

INTRODUCTION **1**

AIMS AND OBJECTIVES **5**

REVIEW OF LITERATURE **7**

MATERIAL AND METHODS **72**

RESULTS AND ANALYSIS **76**

DISCUSSION **96**

CONCLUSION **101**

BIBLIOGRAPHY **102**

PROFORMA **109**

INFORMED CONSENT **111**

ETHICAL CLEARANCE **115**

PLAGARISM CERTIFICATE **116**

MASTER CHART **118**

INTRODUCTION

INTRODUCTION

Malnutrition is a frequent concomitant of surgical illness. Studies have reported 40%-50% of surgical patients to be malnourished on admission to hospital. Malnutrition is a condition that results from eating a diet which does not supply a healthy amount of one or more nutrients. This includes diets that have too little nutrients or so many that the diet causes health problems. The adverse effects of malnutrition on the morbidity and mortality of patients were first recognized by Hippocrates (460 BC - 370 BC) many centuries ago. It is common and occurs in about 30% of surgical patients with gastrointestinal diseases and in up to 60% of those in whom hospital stay has been prolonged because of postoperative complication.[1] There is substantial evidence to show that patients who have signs of malnutrition have a higher risk of complications and an increased risk of death in comparison with patients who have adequate nutritional reserves.[2]

There is substantial evidence to show that patients who have signs of malnutrition have a higher risk of complications and an increased risk of death in comparison with patients who have adequate nutritional reserves.[3] Nutritional assessment is essential for identifying patients who are at risk of developing complications related to significant malnutrition.

A dietary history, physical examination (including anthropometric measurements), and relevant laboratory finding are the appropriate tools needed for an accurate evaluation of a patient's preoperative nutritional status.

The serum albumin level is the most readily available and clinically useful parameter. Serum albumin level greater than 3.5g% suggests adequate protein stores and it confers a protective effect through several biological mechanisms. It predicts perioperative morbidity and mortality. Serum albumin is the most important laboratory test for the diagnosis of protein-calorie malnutrition. Most patients with severe protein depletion will have low serum albumin levels. Patients with abnormal parameter have a markedly increased risk of poor clinical outcomes.[4]

Protein energy malnutrition occurs as a result of relative and absolute deficiency of energy and protein. It may be primary, due to inadequate food intake, or secondary, as a result of other illness.[4] For most developing nations, Primary PEM remains among the most significant health problems. PEM affects every organ system. The most obvious results are loss

of body weight, adipose stores and skeletal muscle mass. Hepatic synthesis of serum protein decreases and depressed levels of circulating proteins are observed. Due to changes in immunological function, wound healing is poor.[4]

Hypoalbuminemia has been shown to be associated with increased mortality and morbidity rates in both hospitalised patients and community dwelling elderly persons. In surgery, an association between hypoalbuminemia and adverse outcome has been recognised for many years. It is common and occurs in about 30% of surgical patients with GI diseases and in up to 60% of those in whom hospital stay has been prolonged because of post-operative complications.[4]

Patients with malnutrition have a higher risk of complications and an increased risk of death. A dietary history, physical examination (including anthropometric measurements), and relevant laboratory investigations are required to know a patient's pre-operative nutritional status.[5]

The serum albumin level is the most readily available and clinically useful parameter. A serum albumin level >3 gm/dL suggests adequate protein stores. It predicts perioperative morbidity and mortality. Serum albumin is the most important laboratory test for the diagnosis of protein-calorie undernutrition. Most patients with severe protein depletion have low serum albumin levels that would correspond to poor clinical outcomes.[5]

The major risk factors that have been commonly studied in relation to the adverse post-operative outcomes in laparotomy patients are intra-abdominal sepsis, old age, obesity, co-morbidities like Diabetes Mellitus, Coronary Artery Disease, Jaundice and Pulmonary Diseases.[5]

Serum albumin is a good indicator of presence and degree of malnutrition and thus an indirect indicator of surgical risk. Hypoalbuminemia causes delayed recovery of bowel function and thus is strongly associated with post-operative complications after surgeries for colonic pathologies and other major GI surgery. The role of albumin in maintenance of homoeostasis is well known but the mechanism of its deficiency and the harmful effects of the same in critically ill patients especially who have undergone major surgery like laparotomies have not been well understood till now. In hypoalbuminemic patients, wound infection, remote infections (pneumonia, anastomotic leakage), are commonly found.[5]

Current indication for nutritional support before elective surgery include a history of weight loss in excess of 10% of body weight or an anticipated prolonged post-operative recovery period during which the patient will not be fed orally.[5]

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

AIM

The aim of study is to find out the role of pre-operative serum albumin as a predictor of post- operative morbidity and mortality in major general surgeries.

OBJECTIVES

- To study the nutritional status of the patient by Serum Albumin level.
- To determine the correlation between pre-operative serum albumin and post-operative morbidity and mortality in major general surgeries.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Physiology of albumin

Human serum albumin is a protein of 585 amino acids with a relatively low molecular weight of 66 kD. It is a simple monomeric protein without prosthetic groups or carbohydrates. Albumin is the most abundant plasma protein accounting for 50-60% of the total plasma protein. The protein is highly soluble as a result of a multitude of acidic (98 Glu + Asp) and basic residues (83 Lys + Arg) and is physiologically negatively charged (-15) due to the uneven content of these amino acid residues.[6]

Structure

Crystallographically, it has been determined that albumin is a polypeptide chain with a heart shaped structure, of which 67% of the configuration, is composed of alpha helices.[7] The protein has dimensions of 80×80×80 Å and a thickness of 30 Å and can be divided into three homologous domains (I-III), that each hold two subdomains (A and B). Except for Cys34, the other 34 cysteine residues form disulphide bridges (seventeen in total).[8]

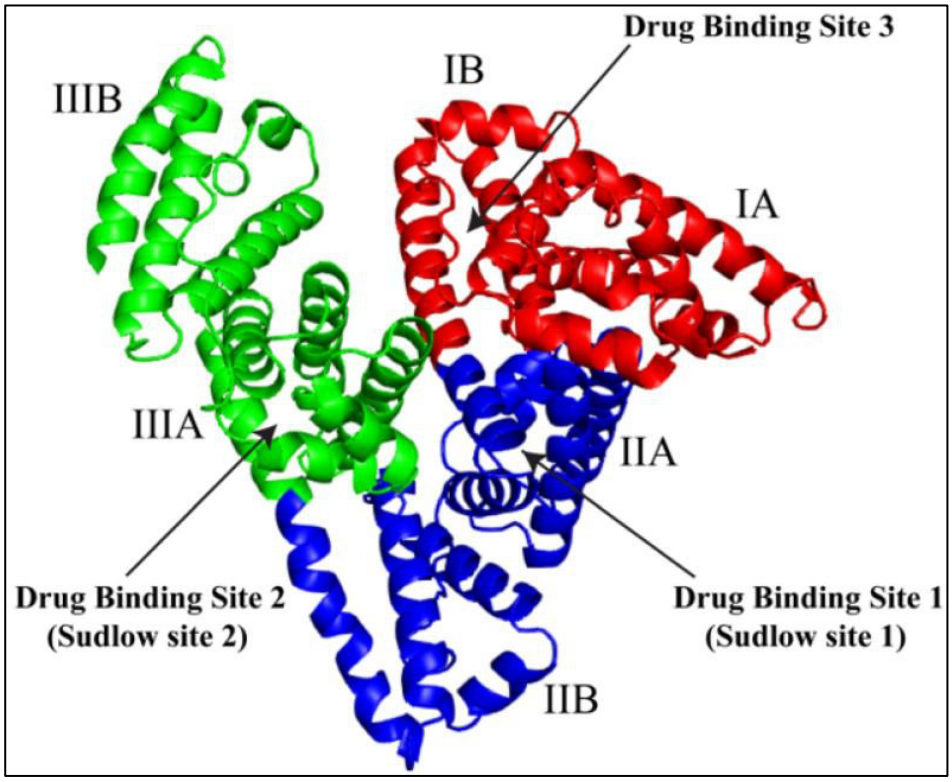


Figure 1 3D MODEL OF A MOLECULE OF ALBUMIN

Synthesis

The albumin genes are located on chromosome 4, at position 4q11−13, close to the centromere for the long arm. The gene consists of a total of 16, 961 nucleotides, counted from the cap site to the first poly(A) addition site. The mRNA encodes for pre-pro-albumin (a precursor protein of 608 amino acids), synthesized by the polyribosomes bound to the endoplasmic reticulum in hepatocytes.[6] The 18 amino acids long N-terminal segment of pre-pro-albumin leads the nascent albumin into the intracisternal space. While the rest of the polypeptide chain is growing, this pre-peptide segment is cleaved off, resulting in a precursor protein of 591 amino acids long, pro-albumin. Afterwards, pro-albumin, the principal intracellular form of albumin, is transported to the smooth endoplasmic reticulum to the Golgi apparatus, where the N-terminal pro-peptide extension of 6 amino acids is removed, completing the synthesis of albumin.[7] Finally, it is secreted into the space of Disse and the hepatic sinusoid. From the start of formation, it takes about 15-30 min for albumin, a non-high priority protein, to appear in the bloodstream and the process of assembling requires energy. Human serum albumin is rapidly and continuously secreted into the bloodstream without being stored. Release on demand from reserves are therefore not possible. A small fraction (< 0,5 %) of the newly formed mature albumin, travels to the hepatic lymph instead.[8]

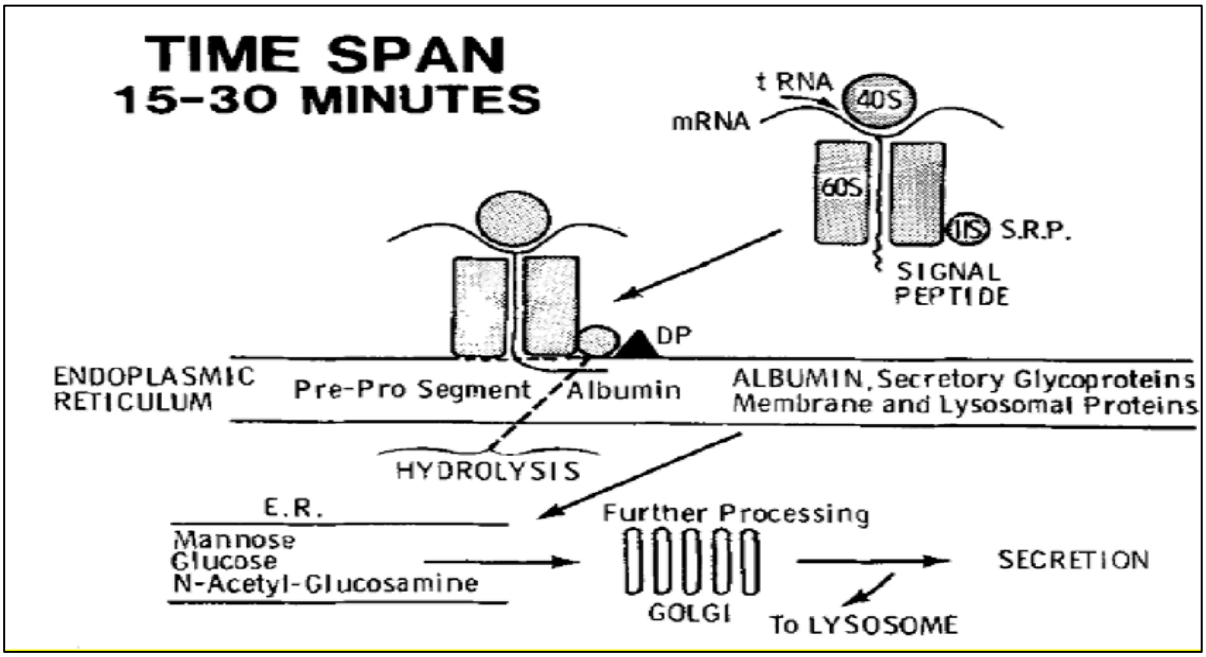


Figure 2 SYNTHESIS OF ALBMIN

Hepatic synthesis of albumin

The liver is the single source of albumin, of which the average synthesis adds up to 9-12 g per day in a healthy individual of around 70 kg. This amount makes up about 25% of the total hepatic protein synthesis. However only 20-30 % of the hepatocytes assemble human serum albumin under physiological circumstances leaving room for regulation of synthesis. Namely, a capacity of a two-to-three-fold increase in albumin synthesis on demand. The physiological regulation is mainly mediated by changes in colloid osmotic pressure in the extravascular hepatic space.[7]

Distribution

The distribution of the total body albumin (+/- 280g in a 70-75 kg individual) can be defined by a two-compartment model. 40% (120g) resides within the intravascular compartment, where the concentration of HSA is about 3,5 -5 g/dl under normal circumstance.[9] The interstitial albumin mass (160g) corresponds to 60% of the total body albumin, making the protein a predominantly extravascular protein regarding mass. However, the extravascular albumin concentration is lower, approximately 60-70% of the plasma albumin concentration. Extravascular albumin distributes to the interstitial compartments of the whole body, but especially the skin and muscle, containing respectively 41% and 40% of the extravascular HSA pool. Albumin is not fixed inside these two compartments (except for 27% of the extravascular albumin pool which is tissue bound). It is exchangeable, meaning it is repeatedly shuttling between the intravascular and extravascular compartments before the protein is broken down.[10] After albumin is secreted from the liver, it circulates in the bloodstream with a circulation half-life of about 16h. Afterwards, it moves across the fenestrated capillary wall into the interstitial space at a rate of 5% per hour of the intravascular albumin content. This is called the transcapillary escape rate (TER), which depends on the capillary integrity and can vary in non-physiological circumstances. These sinusoidal capillaries that allow albumin to pass are mainly located in the liver and spleen, as well as in the lymph nodes, bone marrow and adrenal glands. Besides the transcapillary escape into the extravascular compartment, transcellular movement of albumin is possible in other situations, thus mediated by a receptor (gp60, FcRn). The protein returns from the extravascular space to the plasma through the lymphatic drainage at a rate of 5%/hour, an equivalent to the

transcapillary escape rate. During its lifetime, it moves in and out of the lymphatic system about 28 times.[9]

Catabolism

A steady state exists between albumin synthesis and catabolism under normal circumstances, meaning that about the same amount of albumin, 9-12g, is metabolized as it is synthesized. Serum albumin has a half-life of about 17-19 days, relatively long compared to other plasma proteins. In contrast to the synthesis of albumin which happens in the liver exclusively, the breakdown happens in several organs. 40-60% occurs in the muscle and skin, 10-15% in the liver, 10% in the kidneys and the rest in other places like bone marrow, endothelium, etc. Albumin is degraded by uptake into the endocytotic vesicles located on the endothelial surface. Amino acids are the final product. Furthermore, albumin leaks into the gut (about 1g/d), where the amino acids and peptides are released and reabsorbed. Urinary loss occurs also, but is very minimal with only a few milligrams a day[8–10].

Functions

1) Colloid osmotic pressure

Albumin is the main agent of fluid distribution because of its oncotic properties, as it contributes to 80% of the total intravascular colloid osmotic pressure (25-33 mm Hg), which is crucial in preventing oedema by maintaining a balance between hydrostatic and colloid osmotic pressure inside the vasculature. The osmotic pressure on one side of a semi-permeable membrane is described by the Van't Hoff's equation.[11]

$$\frac{d}{dT}\ln K_{\text{eq}} = \frac{\Delta_r H^{\ominus}}{RT^2}$$

According to the equation, the osmotic pressure of a substance, albumin in this case, is proportional to its concentration and inversely proportional to its molecular mass. Thus, albumin, having a low molecular mass compared to other plasma proteins like gamma globulin, plays a big role in the plasma oncotic pressure. Its low molecular weight accounts for 2/3 of its plasma oncotic function. However, the most relevant factor in the fluid shifts, is the colloid osmotic pressure gradient that is present across the capillary membrane, rather than absolute intravascular or interstitial values.[11]

The amount of fluid flowing through the capillary membrane is described by the Starling equation[11] = $Jv = L\ [(Pc - Pi) - \sigma\ (\pi c - \pi i)]$

Jv = the trans endothelial solvent filtration volume per second

Pc= the capillary hydrostatic pressure

Pi = the interstitial hydrostatic pressure

πc= the capillary protein oncotic pressure

πi = the interstitial oncotic pressure

Lp = the hydraulic conductivity of the membrane

σ = the Staverman's reflection coefficient

The net balance between the hydrostatic pressure and oncotic pressure will decide the movement of fluids. As mentioned, albumin is the main plasma protein influencing the oncotic pressure. Fluid leaves the capillary if Jv is positive and enters the capillary when negative. The other 1/3 of albumin's oncotic functions derives from the Gibbs-Donnan effect: The intravascular albumins that cannot move across the membrane attract positively charged molecules like Na+ and thus, water, due to the global negative charge of albumin at physiological pH. Because albumins are not equally distributed over the interstitial and intravascular compartments, the intravascular albumin compartment, higher in concentration, attracts more molecules, resulting in an influx of osmotically active cations into the intravascular compartment.[9,11]

2) Anti-oxidant activities

Albumin has considerable anti-oxidant properties, that are useful when the body is in a state of oxidative stress, an important factor in a wide range of diseases. When free radicals are produced in abundance, they can inflict damage to sensitive macromolecules. Firstly, albumin can act as a primary antioxidant, that is, it inhibits the formation of NOS and RNS, by way of binding metal ions, such as copper and cobalt. These transition metal ions are involved in the formation of free radicals (Fenton reaction), but when bound to the protein, are not able to do so. Further, albumin can also function as a secondary antioxidant, as it owns a great amount of

reduced sulfhydryl groups, located at the free thiol group, Cys34, that act as scavengers of free radicals (like hypochlorous acid and nitric oxide).[10,11]

3) Ligand binding

Albumin can exercise a transporting function to a variety of compounds by binding to ligands due to the flexibility of albumin's structure. Endogenous substances include bilirubin, fatty acids, hormones, metals and other ions. Drugs are being transported by albumin as well and consequently, their pharmacokinetics are dependent on this binding. Specifically, the binding results in a reduction in the free concentration and thus, restricts the biological activity and distribution of pharmaceuticals.[11]

4) Enzymatic properties

Albumin is capable to take on an enzymatic function, mainly activities engaging in hydrolysis. For instance, the esterase-like (involving Tyr411 or Lys199) and thioesterase activities (of Cys34). These properties can be clinically and pharmaceutically valuable, such as the activation of prodrugs, attained by the esterase activity of albumin.[11]

5) Anti-inflammatory

Because of the ability to modulate the signalling between inflammatory cells, like neutrophils and endothelial cells, albumin can hinder the release of pro-inflammatory cytokines (TNF-α and IFN), generating an anti- inflammatory effect as a result.[11]

6) Anti-thrombotic

Albumin can inhibit platelet aggregation, due to the ability to bind NO to the Cys-34 site, forming nitroso-albumin, which prevents NO to be inactivated quickly, so that NO's anti-aggregant functions are preserved. The protein exerts an anti-coagulant action as well, much like heparin, by augmenting the neutralization of factor Xa by antithrombin[11].

7) Microvascular integrity

The protein contributes to the integrity of microvasculature, directly by binding to the endothelial cells, repulsing other negatively charged molecules and also lowering the permeability of these membranes to large molecules. On the other hand, albumin stabilizes the vessels through its anti-oxidant and anti-inflammatory functions[11].

8) **Plasma buffer**

Albumin possesses 16 histidine imidazole residues that exert a buffering function and thereby are responsible for maintaining physiological plasma pH levels[11].

Hypoalbuminemia

Hypoalbuminemia is defined as a decreased intravascular albumin level of <3.5 g/ml and occurs in various pathological conditions such as sepsis, infection, trauma, nephrotic syndrome, protein-losing enteropathy, trauma, burns, malnutrition, etc. Since there are several factors that play a role in maintaining the homeostasis of serum albumin, as mentioned above, multiple elements contribute to a drop in serum albumin levels in these pathological circumstances: reduced hepatic synthesis, increased catabolism, increased exogenous loss from surgical wounds, increased intravascular permeability, change in lymphatic return of albumin and haemodilution. It is known that a major surgery leads to a postoperative hypoalbuminemia due to a combination of these elements, some contributing more to the drop than others. Elective surgery in the abdominal cavity, in particular, results in a greater decrease in serum albumin, compared to other surgeries.[12,13]

Albumin as a nutritional parameter

Albumin is often used to assess nutrition. Pre-operative screening of nutrition happens frequently as poor nutrition status is a risk factor for postoperative complications. Because of albumin's ease to measure, low cost, reproducibility and consistent response, it is seen as a very efficient screening tool. However, albumin is a very highly criticized nutritional marker. Insufficient dietary protein intake results in a decrease of albumin synthesis, because adequate nutrient intake is necessary for preservation of cellular RNA levels. In acute situations, this has no impact on albumin levels because of the large pool size and low turnover rate. In chronic malnutrition also, studies have shown that a prolonged protein-calorie restriction in healthy patients leads to a decrease in body weight a somatic protein, but little to no decrease in serum albumin due to transport of extravascular albumin to the intravascular compartment as a compensatory measure. As previously stated, the concentration of serum albumin is the result of many events like synthesis, degradation, albumin losses, transcapillary rate and the volume in which albumin resides. Hypoalbuminemia occurs frequently in hospitalized patients because of the inflammatory stress that has an influence on these events. Vascular permeability

especially can increase dramatically in acute settings and cause a significant decrease in albumin level. Various underlying conditions like cancer, hepatic failure, infection, burns, fluid overload and nephrotic syndrome can also be the cause of a hypoalbuminemia. Because of the above-mentioned reasons, serum albumin as laboratory marker on its own, is not an accurate way of assessing nutritional status. It detects an inflammatory state rather than malnutrition. Although hypoalbuminemia can be an indication of malnutrition, to determine a patient's nutritional status, a single lab parameter like serum albumin is not sufficient and should be combined with other assessments like physical examination.[14–16]

Hypoalbuminemia in cancer patients

When discussing postoperative hypoalbuminemia, it is important to be aware of the low albumin level that is already present among some patients that have an underlying condition, cancer for instance. It is known that there is a high prevalence of hypoalbuminemia in cancer patients. In 1954, Babson et al[17] demonstrated that plasma proteins, albumin for instance, could be a nutritional source for tumours by trapping them and using their degradations products for proliferation, which has been confirmed by later studies. Still, the exact mechanism for hypoalbuminemia in cancer patients is not exactly clear, but various theories have been put forward. In the beginning stages the production is enhanced to compensate the low body weight in a cancer patient, but in the later stages, synthesis is suppressed because of inflammation and malnutrition. The body is in a state of physiological stress and responds to the cancer with a release of pro-inflammatory cytokines and growth factors, as part of a systematic inflammatory response. Cytokines, such as IL-6, modulate the synthesis of albumin by hepatocytes. Additionally, the release of tumour necrosis factor could result in an increase of vascular permeability, which leads to albumin moving to the extravascular compartment and causing a low serum albumin level. Serum albumin has been shown to be useful in determining the nutritional status, the severity of the disease, the progression and an independent prognostic factor for mortality in various cancers.[16,17]

POSTOPERATIVE HYPOALBUMINEMIA

CAUSES OF HYPOALBUMINEMIA AFTER ABDOMINAL SURGERY

1) Redistribution

A major surgery puts the body in a state of inflammation and elicits a systematic acute phase response, in which albumin is a negative acute phase protein, a protein which decreases in concentration after an injury. Damaged tissue results in the stimulation of leucocytes, macrophages and mast cells, which in turn causes the release of cytokines such as TNF, HSF, IL-1, IL-2, IL-6 and IFN-γ. These inflammatory mediators, amongst other factors like complement factors C3a and C5a and the activation of the clotting cascade, induce an increase in vascular permeability. The transcapillary escape rate can reach a percentage twice as high as its original rate. The redistribution of albumin from the intravascular to the extravascular space is the most significant contributing factor to the decrease in serum albumin levels that are detected after an elective surgery.[15]

2) Overhydration

Another important element is the peri-operative infusion of intravenous fluids causing an overhydration in healthy subjects. It can hemodynamically result in a reduction in the serum albumin levels because of the direct influence on plasma volume. Besides the hemodynamical influence, overhydration can take a role in enhancing the leakage of albumin, thanks to the fluids' "wash-out" effect of the endothelial surface layer, when delivered in large volumes in a short period of time, increasing the TER.[15]

3) Turnover

The above mentioned systematic acute phase response results in a decreased albumin synthesis as well. Inflammatory mediators, TNF and IL-16, released within this response, make the liver go through a 'hepatic reprioritisation', which means that amino acids are saved for positive acute phase proteins like C-reactive protein, fibrinogen and alpha-2- macroglobulin. These positive acute phase proteins, proteins that increase in concentration after injury, are more important in the host defence process and therefore, their production is facilitated at the expense of hepatically synthesized proteins such as albumin and transferrin. However, this down-regulation of albumin synthesis takes a few hours and thus, cannot be associated to the

rapid postoperative drop in serum albumin. Additionally, the turnover rate is only about 5% per day, meaning reduced synthesis does not a have a major impact on the serum levels. In conclusion, decreased synthesis can have an influence on serum albumin concentration, but not in acute circumstances like these. The degradation rate of albumin, on the other hand, is believed to increase after injury, but is also insufficient and takes a while, to take account for the acute postoperative hypoalbuminemia[15,16].

4) Loss

Surgical wounds and haemorrhage lead to a direct exogenous loss of albumin which can contribute to the decrease in albumin level, but not nearly as significantly as the increased TER.[15]

5) Lymphatic clearance

The lymphatic return of extravascular albumin to the intravascular compartment might be reduced during surgery due to resection of lymph glands in the case of a major abdominal surgery or other elements, like posture, that have an impact on the lymphatic outflow pressure.[15,16]

Hypoalbuminemia as a risk factor after abdominal surgery

It is established that a low serum albumin level is associated with higher mortality and pour outcomes in any kind of clinical setting and is a marker for the severity of the disease. In the surgical patient population, hypoalbuminemia has been linked to important surgical outcomes as well as mortality. A low pre-operative albumin level is proven to be an independent predictive factor of postoperative morbidity and mortality. Post-operative hypoalbuminemia occurring after a surgery is also known to be inversely correlated to various postoperative outcomes, such as mortality, length of stay, surgical site infections, AKI, etc. For this reason, it is important for surgeons to be aware of the decrease of albumin in the early postoperative period, even with normal preoperative levels. Therefore, postoperative hypoalbuminemia and its severity correlates to the severity of the underlying traumatic insult, which correlates with outcome. Albumin reflects this stress response more accurately than other plasma proteins like CRP, because its decrease occurs earlier after surgery than the increase of the CRP level, making it a better predicting factor for postoperative outcome.[12,13,15,16]

Clinical use of albumin

Albumin has been used for more than 60 years and its role in clinical settings has been of keen interest to many medical practitioners. Before papers of the Cochrane collaboration that seriously critiqued the use of albumin, were published, its use was widespread and considered obviously beneficial. After this practice was heavily challenged, medical practitioners had to reconsider the custom of albumin administration which led to this treatment being recommended less and less, other than some specific situations. Even so, albumin is still widely administered in clinical practice mainly for correction of hypovolemia and hypoalbuminemia, despite many findings against a real benefit. The expensive plasma substitute is used for fluid resuscitation in critically ill patients despite many evidences that there is no additional advantage over crystalloids or other colloids, not in terms of mortality or morbidity. Peri-operative albumin resuscitation has also shown no benefit compared to other volume substitutes. Therefore, based on scientific findings, albumin and the other solutions should be considered equivalents.[18,19] Albumin, used to correct hypoalbuminemia in context of surgery or intensive care, is a controversial practice because of the unclear evidence of whether albumin supplementation beneficially influences outcome or not. Other clinical settings where albumin is supplemented are burns, trauma, sepsis, acute respiratory distress syndrome, haemodialysis, ascites, nephrotic syndrome, graft-versus-host disease, cerebral ischemia, enteric diseases, etc. However, most of these indications are not scientifically supported as well. There are some indications where albumin could benefit the clinical outcome.[18] Volume replacement in burn injured patients for example could be a potential indication, for using albumin, but this remains unclear. Either way early use of albumin is not supported. There is some evidence that in cirrhotic patients with ascites, albumin administration could result in more advantageous outcomes. Finally, cardiac surgery, has been considered to be an appropriate indication for using albumin. There has been a debate on whether albumin is considered safe to use. In 1998, a suggestion was put forward by a systematic review that albumin infusion could lead to higher mortality rates. Since then, these claims have been disproven after meta-analyses were published that confirmed the safety of albumin.[20] However, traumatic brain injury is a strong contraindication for resuscitating with albumin, because the use of albumin is proven to be associated with higher mortality rates compared with saline. Attempts have been made to formulate clear guidelines for albumin use, but the question of the appropriate use of albumin remains controversial.[18–20]

NUTRITION IN SURGICAL PATIENT

The primary aim of providing adequate nutrition in patients undergoing surgery is to prevent or reverse the catabolic effects of disease or injury. The efficacy of nutritional regimens has been determined by several important biological parameters but the ultimate substantiation for nutritional support in surgical patients should be improvement in clinical outcome and restoration of function. The incidence of post-operative morbidities, such as intra –abdominal abscess, ileus and anastomotic leakage may be brought down by the use of preoperative nutritional support. Patients with mild to moderate malnutrition need not be supplemented with preoperative nutritional support.[21–23]

Protein depletion results in delayed wound healing. Malnutrition leads to significant mortalities postoperatively and threefold increase in the post operative infection rate. Current indication for nutritional support before elective surgeries include a history of weight loss in excess of 10% of body weight or an anticipated prolonged post operative period of recovery during which the patient will be nil per oral.[24] The outcome of protein calorie malnutrition includes a reduction in lean muscle mass, alteration in respiratory mechanics, impaired immune function and intestinal atrophy. These changes result in increased postoperative morbidity due to impaired wound healing and predisposition to infection.[24–26]

Figure 3 POST-OP EXPLORATORY LAPAROTOMY WITH ANASTOMOTIC LEAK

NUTRITIONAL ASSESSMENT

A comprehensive nutritional assessment comprises of the initial history, physical examination and laboratory testing to provide a snapshot of the patient's recent nutritional health.

1) History

The underlying disease or a history of recent weight loss suggests the possibility of malnutrition. Anorexia, nausea, gastroesophageal reflux, vomiting, dysphagia, or a history of generalized muscle weakness should be investigated for further evaluation. Recent weight loss (5% in the last month or 10% over 6 months) or a current body weight of 80 to 85% (or less) of ideal body weight implies significant malnutrition.[27]

2) Physical Examination

Includes muscle wasting (thenar and temporal muscles), flabby or loose skin (loss of subcutaneous fat), and peripheral oedema and/or ascites (hypoproteinaemia). Hepatomegaly, glossitis, pallor, skin rash, hair changes, gingival lesions and dementia are some of the subtle findings of nutritional deficiency.[27]

3) Anthropometric measurements

Anthropometry is the study that deals with the science of evaluating body size, weight and proportions. The body muscle mass (somatic protein) and fat reserve are reflected by measurements like MHMC (mid humeral muscle circumference) and TSF (triceps skin fold thickness) respectively. Various body compartments (mineral, fat, lean muscle mass) can be assessed by DEXA (Dual-energy X Ray absorptiometry) and it is made available in hospitals. Measurement of weight and height with calculation of BMI can be easily performed in the bedside or at the clinic. BMI is considered to be the most reliable indicator. These values determine the patient's fat reserve with visceral and somatic protein mass.[27]

4) **Laboratory tests**

Serum albumin of less than 3.5 g% in a clinically stable, hydrated patient indicate malnutrition. In dehydrated patients, serum albumin levels are normal in spite of malnutrition. Extravascular compartment contains two thirds of the body albumin stores, remaining one third in the circulation. Plasma levels of albumin are lowered by trauma, sepsis and malignant diseases (catabolic states).[27,28] Serum albumin has 14 -20 days as its half-life.

Serum prealbumin is a more useful indicator of acute change in nutritional status. It has a half-life of 2-3 days. Where,

10-17 mg% - mild depletion

5-10 mg% - moderate depletion

<5mg% - severe depletion

Serum transferrin of less than 200 mg% suggests malnutrition. It has a half-life of 8 -10 days.

5) **Immune function** is altered by malnutrition. It can be assessed by

- Delayed type hypersensitivity
- Total lymphocyte count (TLC) which is calculated by the formula

TLC = %lymphocyte X (WBC /100)

Where, TLC

1500 – 1800 cells/mm – mild depletion

900 – 1500 cells/mm – moderate depletion

<900 cells/mm – severe depletion

NUTRITIONAL INDICES

They provide a means of objective comparison and risk stratification among patients. They aid surgeons in determining the correct timing for intervention and the progress being made towards the goal of adequate nourishment.[28]

1) BODY MASS INDEX (Quetelet's Index)

The Belgian Adolphe Quetelet invented this measure between 1830 and 1850 during the course of developing "Social Physics" 11 It is a statistical measure of the weight of a person scaled in relation to height. BMI is defined as an individual's body weight divided by the square of their height. The extent to which a person's body weight departs can be assessed by calculating BMI from what is normal or desirable for a person of his or her height. The deficiency or weight excess may, in part, be accounted for by body fat or muscle[29]

$$\textbf{BMI} = \textbf{weight (Kg)} / \textbf{height}^2 \textbf{ (m)}$$

= 703 x weight (lb)/ height2(in) BMI correlates with body fat.

BMI raises its advantage in yielding a better estimation of body fat than body weight. BMI correlates with morbidity unlike the ideal body weight tables that were based on mortality data. It is used for both women and men. Mortality and morbidity are correlated with both low and high BMI. Diminished work productivity and lethargy coincides with low levels of BMI. The lowest survivable levels of BMI have been estimated to be 12- 13kg/m2, as suggested by observation in anorexia nervosa, famine and starvation or by theoretical models.[28,29]

Recent studies suggested that the all-cause mortality risk is higher for independently living older individuals with a BMI less than 22 kg/m 2. Owing to low BMI and inadequate dietary intake, the functional status has been diminished among the older individuals in the community. There are few limitations of BMI. It may underestimate body fat in persons who have lost muscle mass (elderly) and may overestimate total body fat in persons who are very muscular (athletes). It will also inaccurately reflect body fat in oedematous states or in individuals who are less than 5 feet tall.[28,29]

			BMI (kg/m2)
Underweight	-	Grade III	<16
		Grade II	16 – 16.99
		Grade I	17 - 18.49
Normal	-		18.5 -24.9
Overweight	-		25-29.9
Obesity	-	Grade I	30 – 34.9
		Grade II	35 – 39.9
		Grade III	>40

BMI is used in children differently. Here it is compared to typical values for other children of the same age but calculated in the same way as for adults. Rather than setting thresholds for underweight and overweight, the BMI percentile allows comparison with children of the same age and sex. A BMI that is less than the 5th percentile is considered underweight, between 85th and 95th percentile at risk of being overweight and above the 95th percentile is considered overweight.[29]

2) PROGNOSTIC NUTRITIONAL INDEX (PNI)

It has been experimented in patients undergoing either major cancer or gastrointestinal surgery and found to identify a subset of patients at increased risk of complications with accuracy. In addition, preoperative nutritional repletion has been shown to reduce post operative morbidity in this patient group.[27,28]

PNI =158 – [16.6 x Alb] – [0.78 x TSF] – [0.2 x TFN] – [5.8 x DH]

Alb – Albumin

TSF – Triceps skin fold thickness (mm)

TFN – Transferrin (mg/dl)

DH – Delayed cutaneous hypersensitivity; induration > 5mm = 2
1 -5mm = 1
Anergy = 0

PNI risk of complication

Low	<40%
Intermediate	40 -49%
High	>50 %

3) NUTRITION RISK INDEX (NRI)

The index uses serum albumin and weight loss as predictors of malnutrition and successfully stratifies perioperative morbidity and mortality. Of note, supplemental nutrition often fails to improve serum albumin levels, hence the NRI is not a tool for tracking the adequacy of nutritional support.[27,28]

NRI = [15.19 x Alb] +41.7 x [actual wt (kg)/ Ideal wt (kg)] NRI

Well-nourished	>100
Mild malnutrition	97.5 -100
Moderate malnutrition	83.5 -97.5
Severe Malnutrition	<83.5

4) CATABOLIC INDEX (CI)

CI = [24 hr urine urea nitrogen excretion in g] – [0.5 x (dietary nitrogen intake in g)]

Where,

No physiologic stress	0
Mild stress	0-5
Moderate to severe stress	>5

ESTIMATION OF CALORIC REQUIREMENTS

Adequate substrates are required for healing and tissue repair. Failure to provide adequate amounts of both calorie and protein leads to further depletion of lean body mass.[27]

A modification of Herring equation has been implicated in the calculation of **Basal Energy Expenditure** (BEE):

BEE in kcal/day in,

Males = 16.4+ [13.7 x weight (kg)] + [5.0 x height(cm)] – [6.8 x age(yr)]

Females = 655 + [9.6 x weight (kg)] + [1.7 x height(cm)] – [4.7 x age(yr)]

The actual caloric requirement is obtained by multiplying BEE by a specific stress factor. Most stressed patients require **25 -30 kcal/kg/day**

TEE in kcal/day (**Total energy expenditure**)

TEE = BEE x stress factor

Selective stress factors

Starvation	0.8 -1.0
Elective surgery	1.0 -1.1
Peritonitis/other infections	1.05 -1.25
ARDS or sepsis	1.3 - 1.35
Cardiopulmonary disease	1.3 – 1.55
Pancreatitis or major burns	1.3-1.8

Estimation of protein requirement

The protein requirement differs according to the clinical condition and also the patient. The appropriate ratio of caloric: nitrogen is 150:1 (for a caloric: protein ratio of 24:1)

METABOLISM OF PROTEINS, CARBOHYDRATES AND FATS

Human body requires energy to maintain itself in a steady state. About 50% of the basal metabolic rate (BMR) represents the work of ion pumping, 30% reflects turnover of proteins and the remaining energy used for recycling of glucose, pyruvate, amino acids and lactate. Energy consumed during physical activity comprises 10 - 20% of the total in hospitalised patients and about 10-50% in normal subjects. The increase in energy expenditure above basal is about 10% for elective surgeries, 10-30% for physical trauma and 50-80% for sepsis. Carbohydrates, proteins and fats are the sources of metabolic energy.[30]

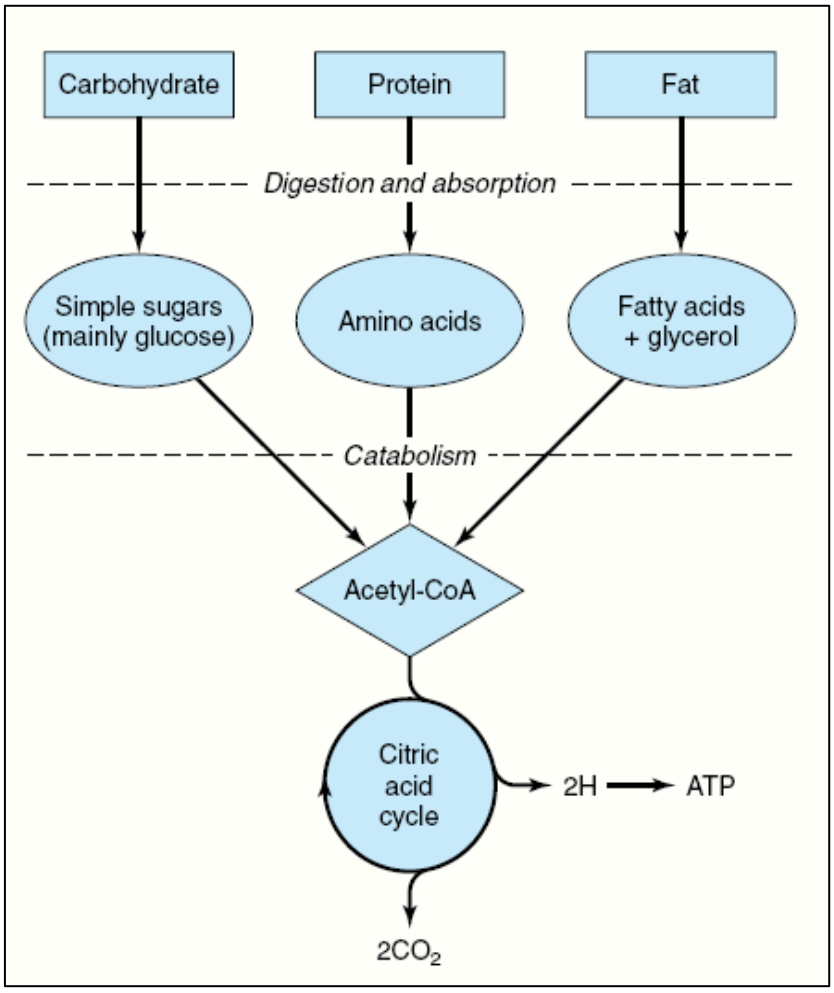


Figure 4 OUTLINE OF THE PATHWAY S FOR THE CATABOLISM OF DIETARY CARBOHYDRATE, PROTEIN, AND FAT

Carbohydrate Metabolism

The body's primary fuel source is constituted by carbohydrates normally that accounts for 30-40% of total caloric intake. Each gram of enteric carbohydrate yields 4 kcal of energy. Parenterally administered carbohydrate (example: IV dextrose) provides 3.4 kcal per gram. Salivary amylase initiates carbohydrate digestion and completion of absorption occurs within 1 - 1.5 m of small intestine. Starch is cleaved by salivary and pancreatic amylases into oligosaccharides. These molecules are hydrolysed and transported by surface oligosaccharides. More than 75% of carbohydrates that are ingested are broken down and absorbed as glucose. Insulin secretion from pancreatic beta cells is stimulated by hyperglycaemia. Insulin then influences protein synthesis. A minimum intake of 400 kcal of carbohydrate per day reduces protein breakdown, especially after adaptation to starvation. Wound healing requires glucose as a major component, but excessive carbohydrate intake or repletion with excess amounts of glucose can result in hepatic steatosis and neutrophil dysfunction. Excess glucose from refeeding, as reflected by RQ >1.0, occurs in conditions such as thermogenesis, glucosuria and conversion to fat (lipogenesis). Excessive glucose administration is deleterious in patients with suboptimal pulmonary function because of its elevated carbon-di-oxide production. It results in hyperglycaemia, which may contribute to increased risk of infection and immune suppression. As a result of carbohydrate deficiency breakdown of muscle tissues will occur. Fatiguability will set in as deficiency evolves. Cerebral metabolism diminishes and thinking capacity is hindered. Deficiency also causes ketosis and ultimately weight loss which on a long run can cause acidosis. In addition, patient may have nausea, mood swings, dizziness, weakness and depression. Hence adequate dietary carbohydrate is mandatory.[31]

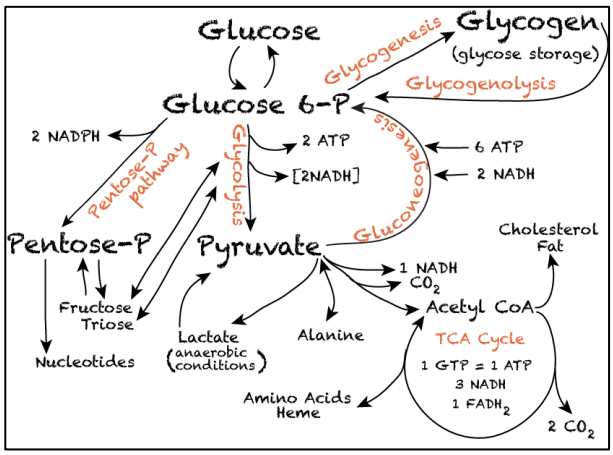


Figure 5 GLUCOSE METABOLISM

Protein metabolism

Amino acids comprise proteins their and metabolism produces 4 Kcal/gm of protein. Single amino acids and dipeptides are produced by protein digestion that are absorbed by the gastrointestinal tract. The process of digestion is initiated by gastric pepsin. Enterokinase, that are the principal effectors of protein degradation are found throughout the duodenal mucosa and activate pancreatic proteases. Once digested, almost 50% of protein is absorbed in duodenum and complete protein absorption occurs in the mid jejunum. A 70 kg person has a total body protein of 10 - 11 kg that is concentrated predominately in skeletal muscle. Daily protein turnover is 3 % of total body protein that accounts to about 250-300 g. The daily protein requirement in healthy adults is 0.8 g/kg body weight.[32,33] Protein synthesis or breakdown can be determined by measuring the nitrogen balance. Nitrogen of 1 g accounts to about 6.25g of protein intake.

Nitrogen (balance) = Nitrogen (intake) – Nitrogen (output)

Nitrogen (intake) = gm protein (intake)/6.25 Nitrogen (output)

= (UUN X Vol) +3

Where, UUN is urine urea nitrogen vol is volume of urine produced over time of measurement. There are 20 amino acids that are divided into essential amino acids and non-essential amino acids, that depends on whether the synthesis is de novo. Major role of amino acids is:

1) Protein synthesis and recycling.
2) Energy generation through catabolic reactions and carbon-di-oxide production.
3) Incorporation of nitrogen into non-essential amino acids and nucleotides.
4) Transport and storage of small molecules and ions.

Plasma amino acid accumulation is regulated primarily by the hepatic metabolism of absorbed amino acids. Urinary excretion amounts to about 10- 15g of basal nitrogen loss. A decrease of protein turnover occurs as age advances with 25g/kg/day in the neonate to 3g/kg/day in the adult.[32,33]

Glutamine

It is a non-essential amino acid that plays a vital role in the metabolically stressed patient. It is considered to be a crucial respiratory fuel for enterocytes. The decline of glutamine associated with injury or stress exceeds that of any other amino acids and persists during recovery even after the concentration of other amino acids has normalized. It is essential for lymphocyte production and macrophage function.[32]

Arginine

It is a substrate for the urea cycle and nitric oxide production. It improves nitrogen balance and wound healing, stimulates T-cell response and reduces the incidence of infective complications. It also improves albumin synthesis.[33]

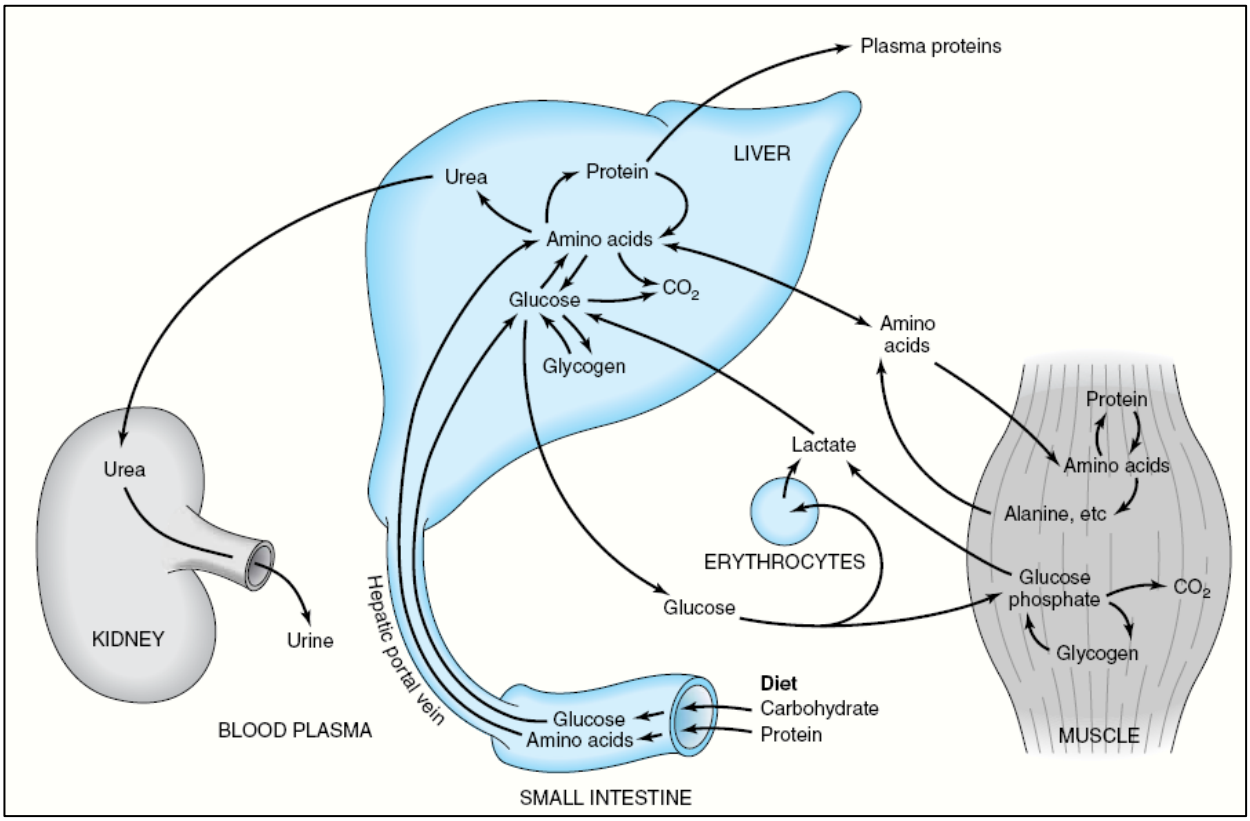


Figure 6 OVERVIEW OF PROTEIN METABOLISM SHOWING MAJOR PATHWAYS AND END PRODUCTS

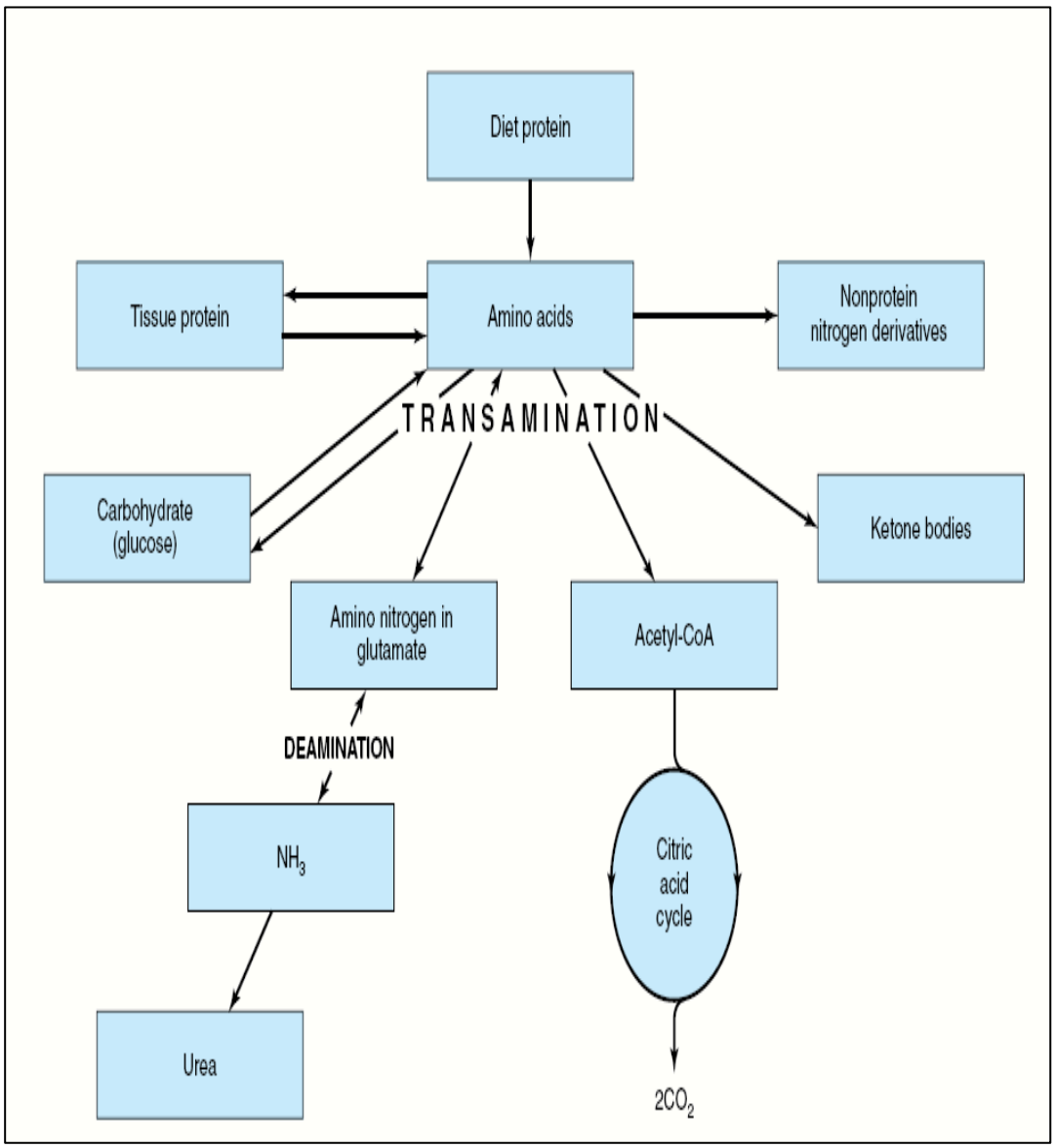


Figure 7 FATE OF DIETARY PROTEIN

Lipid metabolism

About 25 - 40 % of caloric intake in a typical diet is constituted by lipids. 9 kcal of energy is obtained from each gram of lipid. A functional jejunum and ileum along with co-ordination between pancreatic and biliary secretion is required for the complex digestion of lipid molecules. Duodenal introduction of fat results in secretion of secretin and cholecystokinin and these are essential for gall bladder contraction and pancreatic enzyme release, respectively. The hydrolysis of triglycerides to two fatty acids and a monoglyceride by lipase is felicitated by the alkaline environment of the duodenum. Emulsification is due to bile salts. Micelle formation is the most important step in lipid absorption, which helps absorption of fats across the mucosal barrier. The bile salt pool is maintained through reabsorption of bile salts and increased hepatic cholesterol synthesis.[34]

Increased hepatic synthesis of cholesterol compensates the intestinal bile salt losses. Bile salt pool would get depleted and fat malabsorption occurs due to major ileal resection. Glucagon, catecholamines, steroids stimulate lipolysis that is inhibited by insulin. Dramatic lipolysis occurs due to stress. The cell membrane integrity is maintained by essential fatty acids linolenic acid and linoleic acid. The sole precursors of eicosanoid production are dietary fats. Generalized scaling rash, bony changes and hepatic steatosis occurs due its clinical deficiency.[35,36]

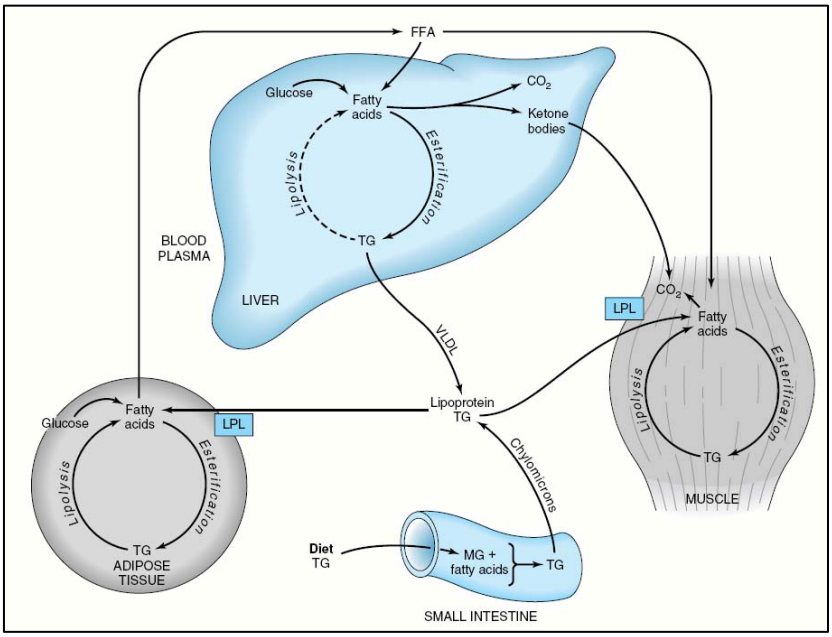


Figure 8 LIPID METABOLISM

Functions of plasma proteins

Function	Plasma Proteins
Antiproteases	Antichymotrypsin α_1-Antitrypsin (α_1-antiproteinase) α_2-Macroglobulin Antithrombin
Blood clotting	Various coagulation factors, fibrinogen
Enzymes	Function in blood, eg, coagulation factors, cholinesterase Leakage from cells or tissues, eg, amino-transferases
Hormones	Erythropoietin[1]
Immune defense	Immunoglobulins, complement proteins, β_2-microglobulin
Involvement in inflammatory responses	Acute phase response proteins (eg, C-reactive protein, α_1-acid glycoprotein [orosomucoid])
Oncofetal	α_1-Fetoprotein (AFP)
Transport or binding proteins	Albumin (various ligands, including bilirubin, free fatty acids, ions [Ca^{2+}], metals [eg, Cu^{2+}, Zn^{2+}], metheme, steroids, other hormones, and a variety of drugs Ceruloplasmin (contains Cu^{2+}; albumin probably more important in physiologic transport of Cu^{2+}) Corticosteroid-binding globulin (transcortin) (binds cortisol) Haptoglobin (binds extracorpuscular hemoglobin) Lipoproteins (chylomicrons, VLDL, LDL, HDL) Hemopexin (binds heme) Retinol-binding protein (binds retinol) Sex hormone-binding globulin (binds testosterone, estradiol) Thyroid-binding globulin (binds T_4, T_3) Transferrin (transport iron) Transthyretin (formerly prealbumin; binds T_4 and forms a complex with retinol-binding protein)

[1]Various other protein hormones circulate in the blood but are not usually designated as plasma proteins. Similarly, ferritin is also found in plasma in small amounts, but it too is not usually characterized as a plasma protein.

Figure 9 FUNCTIONS OF PLASMA PROTEIN

PLASMA PROTEINS

The major solid components of plasma are made up of the total proteins that constitute 7 to 7.5 g/dl. Varied types of lipoproteins and conjugated proteins also include itself along with simple proteins in the complex mixture of plasma proteins.

Though under normal circumstances the number of individual antibodies is low, there are thousands of circulating antibodies. Globulins, albumin and fibrinogen are the three major groups of antibodies.

The most common method used for analysis of plasma proteins is electrophoresis.

ALBUMIN

About 60% of total plasma proteins is constituted by the major protein of human plasma - albumin. The normal serum value is between 3.5-5.5 g/dl. Molecular weight is 69kda. The total exchangeable pool of albumin is 4-5g/kg body wt.; between 6-10% of the exchangeable pool is degraded per day.

Human albumin does not increase the viscosity of plasma due to its ellipsoid shape and consists of 17 disulphide bonds and 585 amino acids arranged in a single polypeptide chain. It accounts for 75-80% of human plasma 's osmotic pressure. Total protein of greater than 5 g/dl and serum albumin of greater than 2.5 g/dl is needed to produce adequate oncotic pressure. Albumin synthesis is decreased during fasting and in cases of protein malnutrition.

Albumin is an acute phase protein the concentration of which is decreased by at least 25% following injury. Time course of changes occurs in some major acute phase proteins[12,13] C3, C3 component of complement.

Extent Of Malnutrition

Normal	-	3.5-5.5gm/dl
Mild	–	2.8-3.5gm/dl
Moderate	-	2.1-2.7gm/dl
Severe	-	<2.1gm/dl

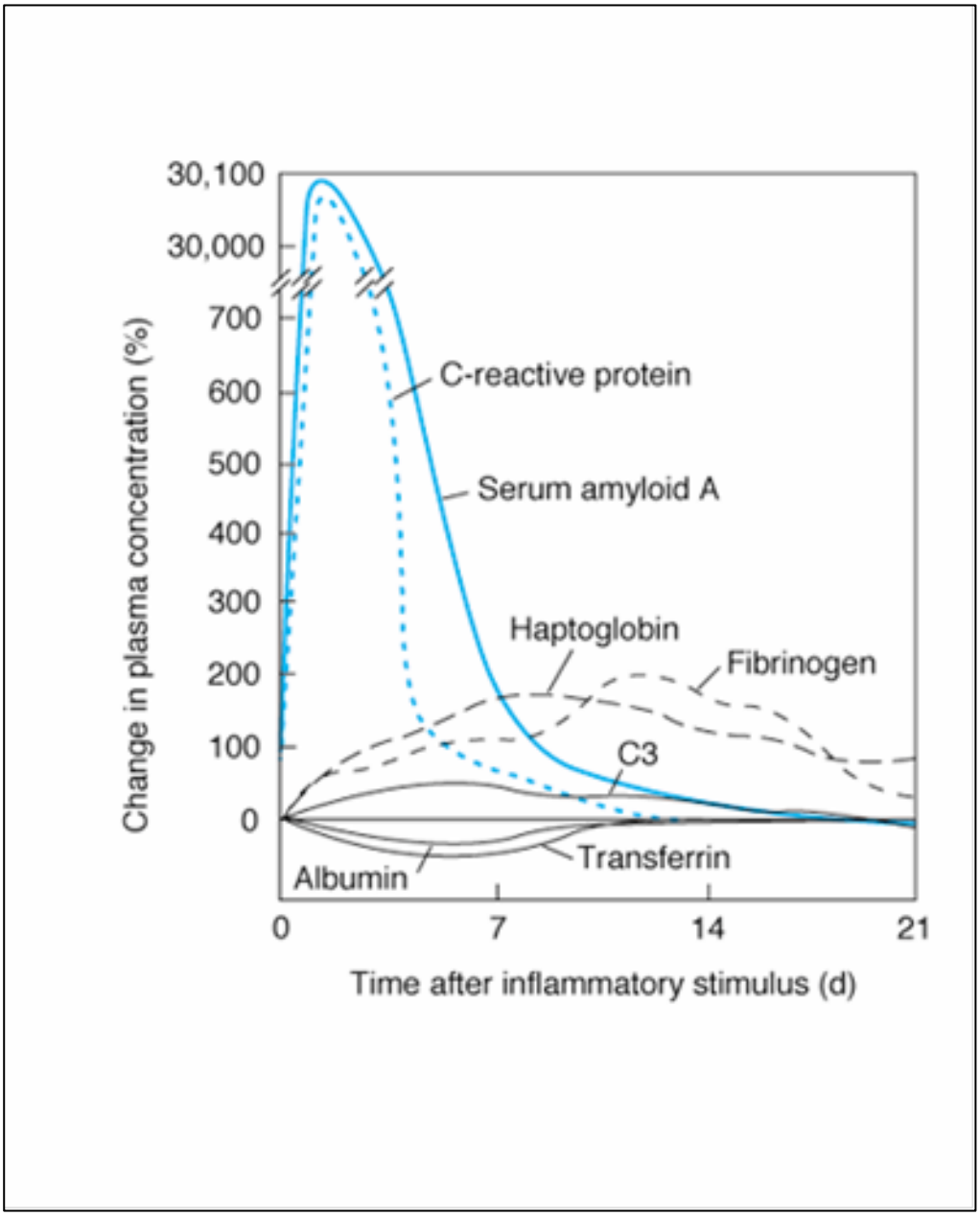


Figure 10 TIME COURSE OF CHANGES IN MAJOR ACUTE PHASE PROTEINS

Causes Of Hyperalbuminemia (>5.5g/dl) - There are no known pathological causes for a raised plasma albumin. Causes include:

1) Dehydration: this is also reflected in an increase in plasma total protein, a raised haematocrit and appropriate clinical features.
2) Venous stasis
3) Albumin infusion

Causes Of Hypoalbuminemia (<3.5g/dl)

1) Increase in plasma H - This occurs as a part of physiological response in pregnancy. Other causes include, excessive infusion of IV fluids, water retention in SIADH, glucocorticoid deficiency.
2) Diminished synthesis - Any cause of generalized protein malnutrition will ultimately be reflected in low plasma albumin. Causes for this include deficient diet in protein nitrogen, protein malabsorption such as coeliac disease, tropical sprue, crohn's disease, cystic fibrosis, decreased synthesis in chronic liver disease and in hereditary analbuminemia where there is a marked impairment of albumin biosynthesis with plasma levels which are typically low.
3) Increased catabolism - This is a feature of hypercatabolic state. The important feature of hypercatabolic state is the stress related stimulation of glucocorticoid production. These hormones are known to stimulate protein catabolism. Condition such as fever, trauma, major surgery, severe sepsis, malignant disease may all be associated with varying degrees of hypoalbuminemia.
4) Losses of albumin from body - Sites of excessive loss are GI tract, kidneys and skin. Hypoalbuminemia correlates itself with immune response impairment such as activation of macrophages and formation of granuloma, decreased wound healing mechanics, and impairment of collagen meshwork synthesis at surgical wounds and anastomotic sites. This signifies the reason why infections such as pneumonia, wound infection and anastomotic site leakage were commonly found among patients with hypoalbuminemia.

STRESS METABOLISM

Physiologic process, immunocompetence, wound healing and recovery from critical illness all depend upon adequate nutrient intake.

STARVATION

Hepatic glycogen is depleted after an overnight fasting due to decreased plasma insulin and a rise in glucagon levels. After a day's fast, carbohydrate stores are depleted. Muscle glycogen is used after hepatic glycogen. Hepatic glucose metabolism must satisfy the energy demands of the hematopoietic and the central nervous system particularly the brain, which is dependent on glucose oxidation during acute starvation. Within approximately 10 days of starvation, the brain adapts to use fat as its fuel source. Thereafter brain relies on ketoacids produced by the liver. This has a protein sparing effect. Relative protein preservation, changes in the fuel consumption, and a decrease in basal expenditure of energy are the adaptive changes to starvation (up to 30%).[37]

Physiologic stress

Sepsis, trauma and major operations resulting in interaction of endocrine and metabolic responses are divided into three phases

Catabolic phase

There is a dramatic increase in the metabolic demand after a major surgery that is reflected by an increase in urinary nitrogen excretion. Following a major surgery, depletion of proteins initially occurs because patients commonly have an elevated basal metabolic rate and they are kept nil per oral. Physiological stress leads to hormonal responses that includes elevation in the serum glucagon, glucocorticoids and catecholamine levels.[37]

Early Anabolic phase (Cortical withdrawal phase)

Since it marks the shift from catabolism to anabolism, it ranges from several weeks to few months. A progressive gain in weight and muscle strength occurs during this phase due to positive nitrogen balance. The amount of energy lost in catabolic phase equals the amount of nitrogen gained.[37]

Late Anabolic phase

This refers to the last period of recovery that lasts for months to several weeks. Nitrogen balance equals as adipose tissue stores are lost gradually. Following elective operation, neural impulses carried via spinothalamic pathways activate the brain stem and thalamic and cortical centres which stimulate the hypothalamus. Hypothalamic stimulation triggers combined neural and endocrine discharges. Nor epinephrine is released from sympathetic nerve endings. Epinephrine from the adrenal medulla, Aldosterone from adrenal cortex, ADH from posterior pituitary, insulin and glucagon from the pancreas and ACTH, TSH and GH from the anterior pituitary. These hormones produce secondary elevations of cortisol, thyroid hormone and somatomedins. The effects of the heightened neuroendocrine secretion include.[37]

1) Peripheral lipolysis from the synergistic activation of hormone sensitive lipase by glucagon, epinephrine, cortisol and thyroid hormone
2) Accelerated catabolism, a rise in proteolysis stimulated by cortisol and
3) Decreased peripheral glucose uptake due to insulin antagonism by GH and epinephrine.

The consequences are a marked rise in plasma concentration of free fatty acids, glycerol, glucose, lactate and amino acids. The liver responds with an increase in substrate uptake and glucose production, as a result of glucagon stimulated glycogenolysis and enhanced gluconeogenesis induced by cortisol and glucagon. Operation and trauma are neuroendocrine driven processes. The BEE rises by 10% in post operative patients.[37]

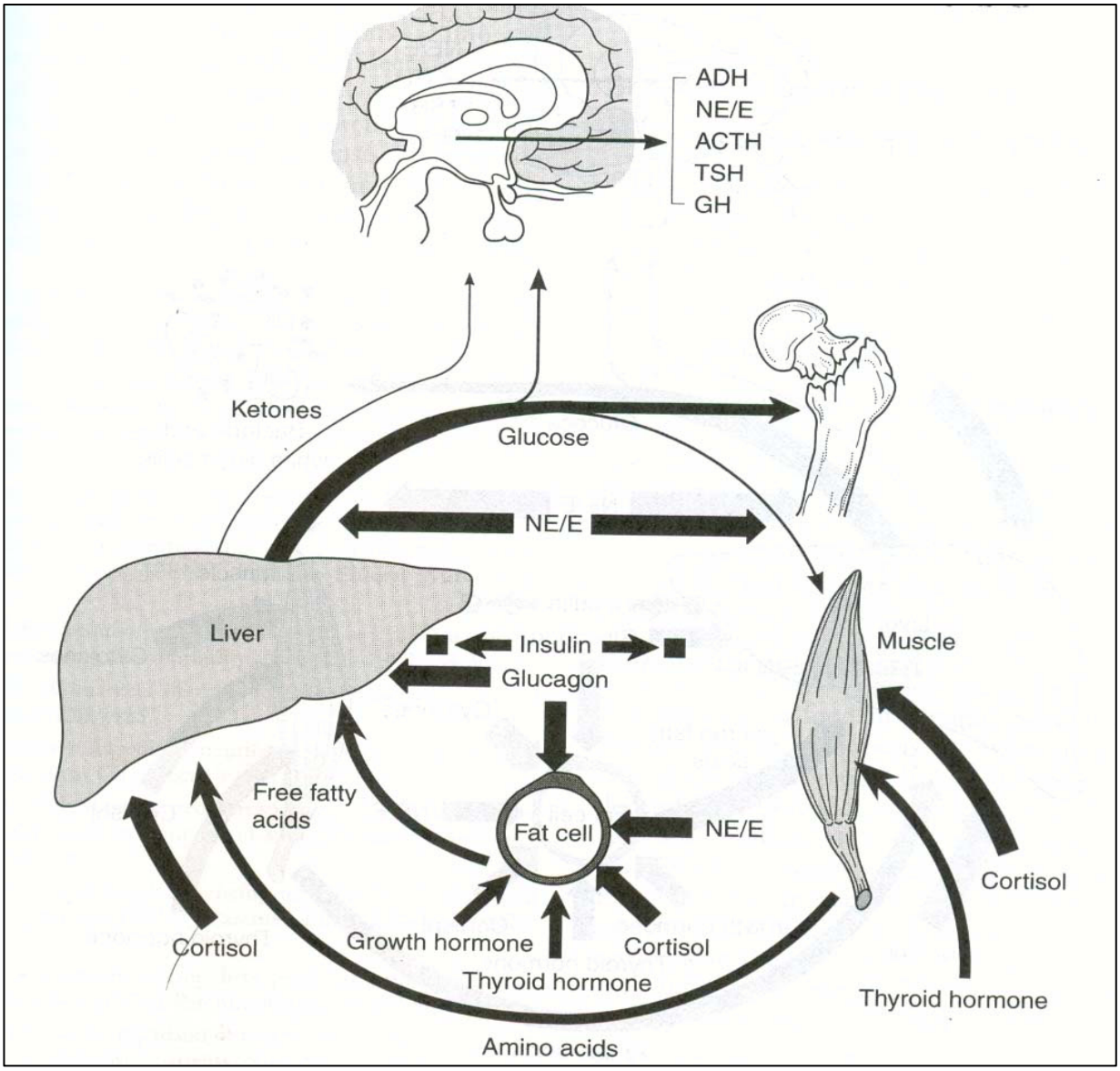


Figure 11 THE METABOLIC RESPONSE TO TRAUMA

WOUND HEALING

Healing is a proliferative reaction that restores tissue integrity. It involves the following processes.

1. Induction of an inflammatory process in response to the initial injury with removal of damaged and dead tissue.
2. Proliferation and migration of parenchymal and connective tissue cells.
3. Formation of new blood vessels (angiogenesis) and granulation tissue.
4. Synthesis of ECM proteins and collagen deposition.
5. Tissue remodelling.
6. Wound contraction.
7. Acquisition of wound strength.[38]

Minimal intervention is required after a typical injury as wound healing per say is a normal orderly process. A normal wound healing state is achieved after a variety of interventions that are required to shift and correct the wound healing process in chronic wound healing. Acute wounds are surgical or traumatic in origin. These wounds occur drastically and as a result of rapid and predictable repair process, results in closure. Chronic wounds arise as a result of repeated insult to tissues, compromise to vasculature or chronic inflammation. They fail to go through the normal repair processes. They result in an untimely closure or improper wound closure.[38]

WOUND HEALING PHASES

1) Inflammation and haemostasis
2) Proliferation
3) Maturation and Remodelling

The immediate response to injury is the inflammatory phase (also called reactive phase). The body's defence system aims to prevent further injury or to limit the amount of damage. The proliferative phase (also called regenerative or reparative phase) consists of re-epithelialisation, matrix synthesis and neovascularisation to relieve ischemic trauma itself. The final phase is maturational (or remodelling) phase which is defined as the period of contraction of the scar with cross linking of the collagen and loss of oedema.[38,39]

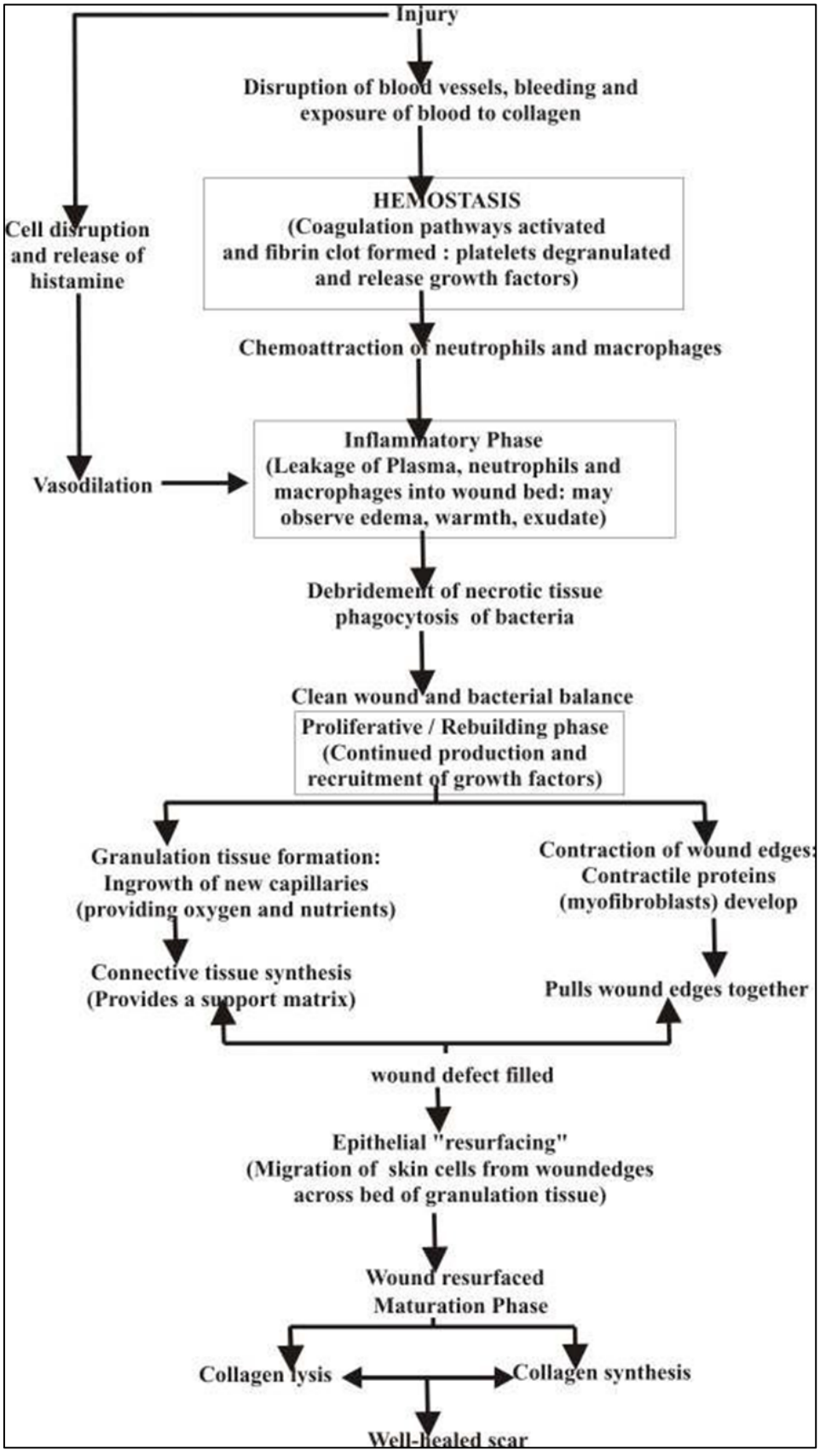

Injury
Disruption of blood vessels, bleeding and exposure of blood to collagen
HEMOSTASIS
(Coagulation pathways activated and fibrin clot formed : platelets degranulated and release growth factors)
Cell disruption and release of histamine
Chemoattraction of neutrophils and macrophages
Inflammatory Phase
(Leakage of Plasma, neutrophils and macrophages into wound bed: may observe edema, warmth, exudate)
Vasodilation
Debridement of necrotic tissue phagocytosis of bacteria
Clean wound and bacterial balance
Proliferative / Rebuilding phase
(Continued production and recruitment of growth factors)
Granulation tissue formation:
Ingrowth of new capillaries
(providing oxygen and nutrients)
Contraction of wound edges:
Contractile proteins
(myofibroblasts) develop
Connective tissue synthesis
(Provides a support matrix)
Pulls wound edges together
wound defect filled
Epithelial "resurfacing"
(Migration of skin cells from woundedges across bed of granulation tissue)
Wound resurfaced
Maturation Phase
Collagen lysis
Collagen synthesis
Well-healed scar

Figure 12 STAGES OF WOUND HEALING

INFLAMATORY PHASE

Inflammation occurs as an immediate reaction of the tissue to injury. Here an attempt is made to limit damage by stopping the bleeding, sealing the surface of the wound and removing any necrotic tissue, foreign debris or bacteria present. During the inflammatory phase, increase in vascular permeability, chemotactic migration of cells, cytokine secretion and growth factor migration to the inflammatory site occurs along with the migratory cell activation.[40]

HEMOSTASIS AND INFLAMMATION

Vascular damage occurs after an acute tissue injury exposing the subendothelial collagen to platelets. This leads to activation of coagulation pathway after the process of platelet aggregation. Initially increase in vascular permeability occurs due to vasodilatation that follows intense arteriolar and capillary vasoconstriction. The platelets and erythrocytes adhere to the damaged capillary endothelium along with capillary plunging, thus ceasing haemorrhage. Architecture as of the pre-existing tissues is achieved by regeneration with absence of scar formation. Regeneration though the goal of wound healing, is found only in early embryonic development or in certain tissues such as liver and bone.[40] However, the accuracy of regeneration is sacrificed for the inflammatory phase in wound healing of adult humans.

PROLIFERATIVE PHASE

Wound repair begins to start as the acute responses such as haemostasis and inflammation resolves through angiogenesis, fibroplasia and epithelialisation. During this phase formation of granulation tissue takes place which consists of hyaluronic acid, fibronectin, loose collagen arrangement, macrophages, fibroblasts and the capillary bed.[40]

ANGIOGENESIS

It is a phenomenon of new vasculature formation. It supports the healing wound environment after injury. The basement membrane of post capillary venules is degraded by the activated endothelial cells, thereby allowing the migration of cells through the gaps. Division of the endothelial cells that migrate takes place resulting in tubule or lumen formation. Eventually capillary maturation occurs due to deposition of the basement membrane.[40]

FIBROPLASIA

In connective tissues, the resting mesenchymal cells are differentiated to form the specialised cells called fibroblasts. Following an injury, the normal fibroblasts that are dormant and scanty divide and produce the components of extracellular matrix, after being chemoattracted to the inflammatory site. The fibroblasts' chief function is considered to be the synthesis of collagen. The time needed for undifferentiated mesenchymal cells to differentiate into highly specialised fibroblasts is the delay between injury and the collagen appearance in the healing wound. This period depends on the type of tissue injured. It generally takes 3 to 5 days, known as the lag phase in the process of wound healing. The collagen synthesis rate declines after 4th week and balances rate of collagen destruction eventually with the aid of collagenase. The wound enters the collagen maturation phase at this point. The maturation phase extends for months and years. During the maturation phase glycoprotein and mucopolysaccharide levels decrease and new capillaries revert and disappear. All these changes increase the wound strength and also alter the appearance of wound.[39,40]

EPITHELIALISATION

Re-epithelialisation of wound begins within hours after injury and takes 48 hours to complete in case of approximated incised wounds. Initially wound is quickly sealed by formation of clot and then by migration of epithelial cells across the defect. Keratinocytes are situated at basal layer of the residual epidermis and also in depths of dermal appendages lined by epithelium. These migrate to re surface the wound. Epithelialisation is characterized by a series of changes in the wound that is detachment, migration and proliferation after which follows the differentiation and later stratification. Epithelialisation proceeds more rapidly only when the basement membrane zone is intact.[40]

EXTRACELLULAR MATRIX

The ECM provides a scaffold for the stabilization of the physical structure of the tissues. Cells within it produces the macromolecular constituent that includes the following:

1) Glycosaminoglycans, or polysaccharide chains are usually found linked covalently to protein in the form of proteoglycans.
2) Fibrous proteins that include collagen, fibronectin, elastin and laminin.

The wound matrix accumulates and changes in composition take place as healing progresses, maintaining a balance between new deposition and degradation. The provisional matrix exists as a scaffold for cellular migration and composed of fibrin, fibrinogen, fibronectin, and vitronectin. Glycosaminoglycans and proteoglycans which are synthesized later aid in matrix deposition and remodelling. Collagen which are the major scar proteins are the final results.[39]

WOUND CONTRACTION

The wounds undergo certain degree of contraction. The myofibroblast is the major cell responsible in this process. These cells start appearing from day 6. Immediately after injury the wound contraction almost begins. The cellular movement with concomitant cytoskeleton reorganization is responsible for the wound contraction.[40]

WOUND STRENGTH

At the 1st week end, the strength of the wound is roughly 10% that of a normal unwounded skin, which quickly increases within next 4 weeks. By the end of 3rd month, a plateau accounting for about 70% to 80% of the normal unwounded skin's tensile strength, is reached. This condition may persist for life. The excess of collagen formation over collagen destruction during the 1st 2 months of healing and at later times from structural modification of collagen fibres (cross linking, increased fibre size) after cessation of collagen synthesis is found to be responsible for the recovery of tensile strength.[40]

This picture clearly depicts the various wound healing phases where the inflammatory phase in the earlier stages progresses to granulation tissue formation and ultimately wound contraction. Subsequently collagen accumulation and remodelling occur.

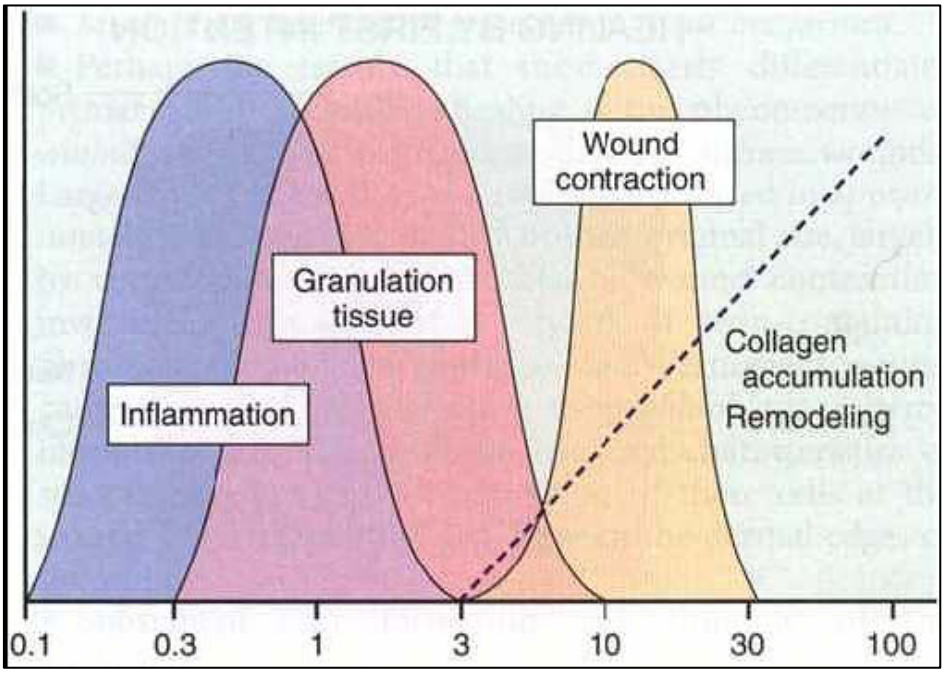
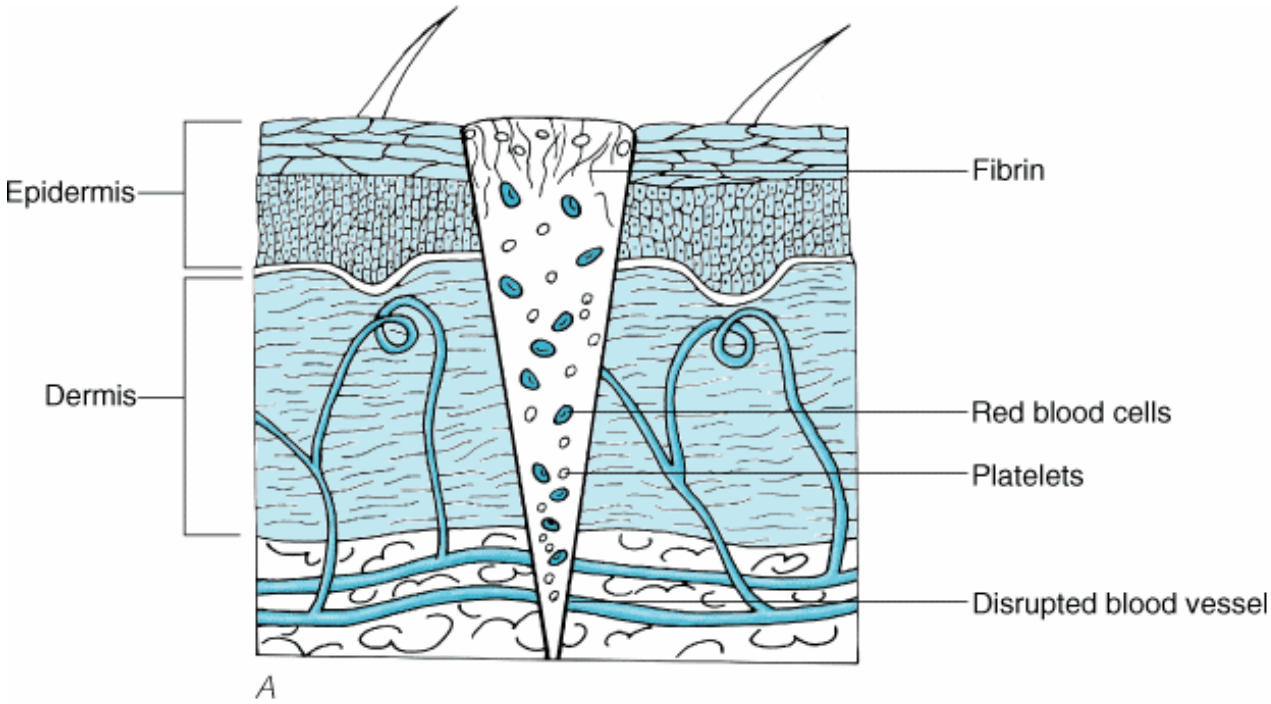


Figure 13 PHASES OF WOUND HEALING



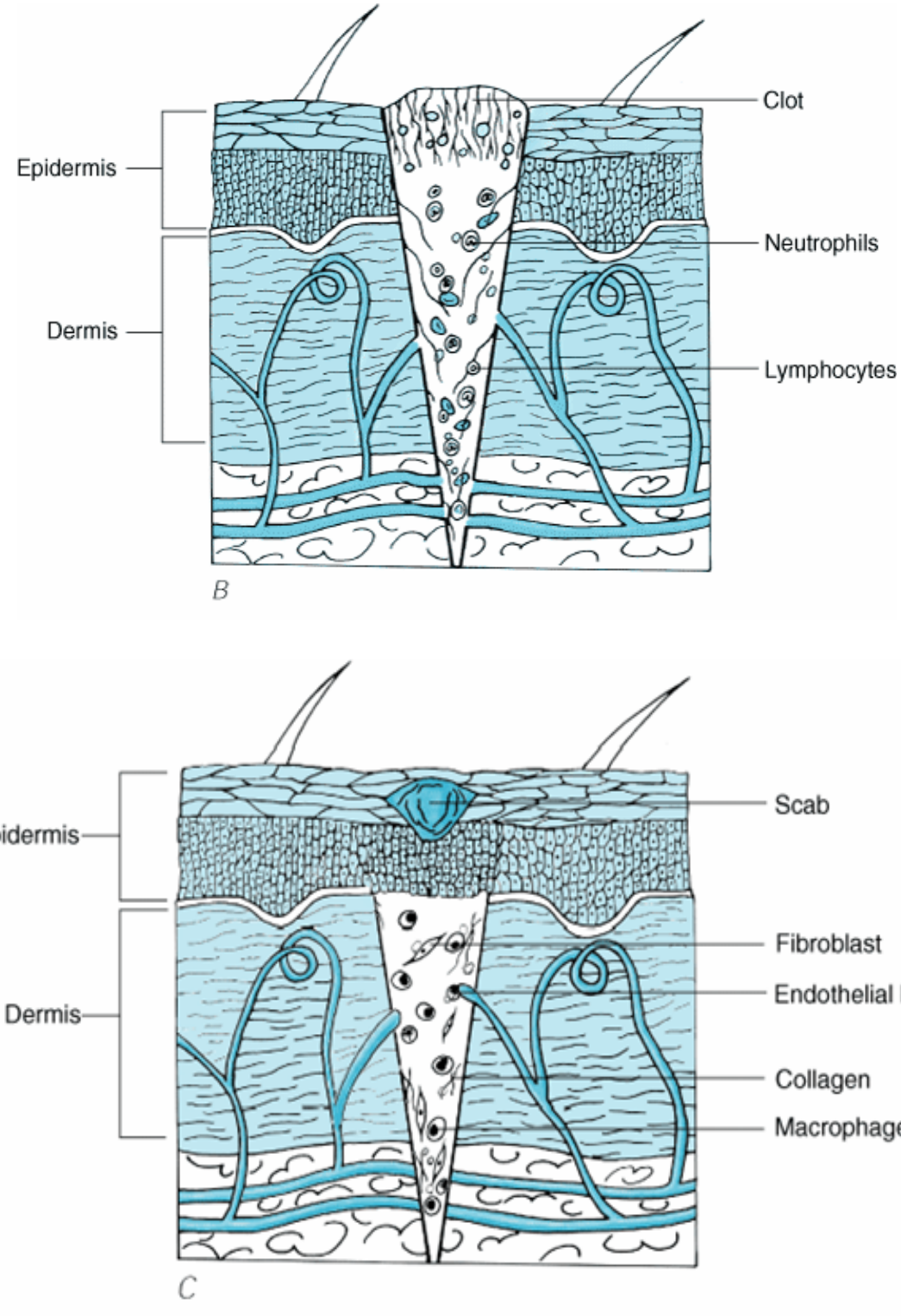


Figure 14 DIAGRAMATIC REPRESENTATION OF STAGES OF WOUND HEALING

The pictorial representatives clearly depict the various histological stages where in platelets start aggregating once the subendothelial collagen matrix is exposed, with subsequent accumulation of lymphocytes and macrophages and the meshwork of collagen, fibroblasts migrate to the wound site and thus aid in healing.[40–42]

Based on timing of wound healing they are classified into:

1) HEALING BY PRIMARY INTENTION

It occurs when the wound is closed by direct approximation of the wound margins or by placement of a graft or flap. Wounds that are less than 6 hrs old are considered and are less likely to develop into chronic wounds.

2) HEALING BY SECONDARY INTENTION

It occurs when a wound is left open and is allowed to close by epithelialization and contraction. It is commonly used in the management of wounds that are treated beyond the initial 6 hrs.

3) HEALING BY TERTIARY INTENTION

It is used for wounds that are heavily contaminated for primary closure but appears clean and well vascularised after 4-5 days of open observation so that the cutaneous edges can be approximated at that time.

ABNORMAL WOUND HEALING

In such a complex series of intervening events as wound healing, multiple factors can impede the outcome affect. The factors that affect the time of recovery are the amount of tissue lost or damaged, the amount of foreign material or bacterial inoculation and the length of exposure to toxins. The greater the insult, the longer the reparative process and the greater the amount of residual scar. Intrinsic factors that affect wound healing are chemotherapeutic agents, atherosclerosis, cardiac or renal failure. The blood supply in the lower extremity is the worst in the body and that on the face and hands is the best.[40,41]

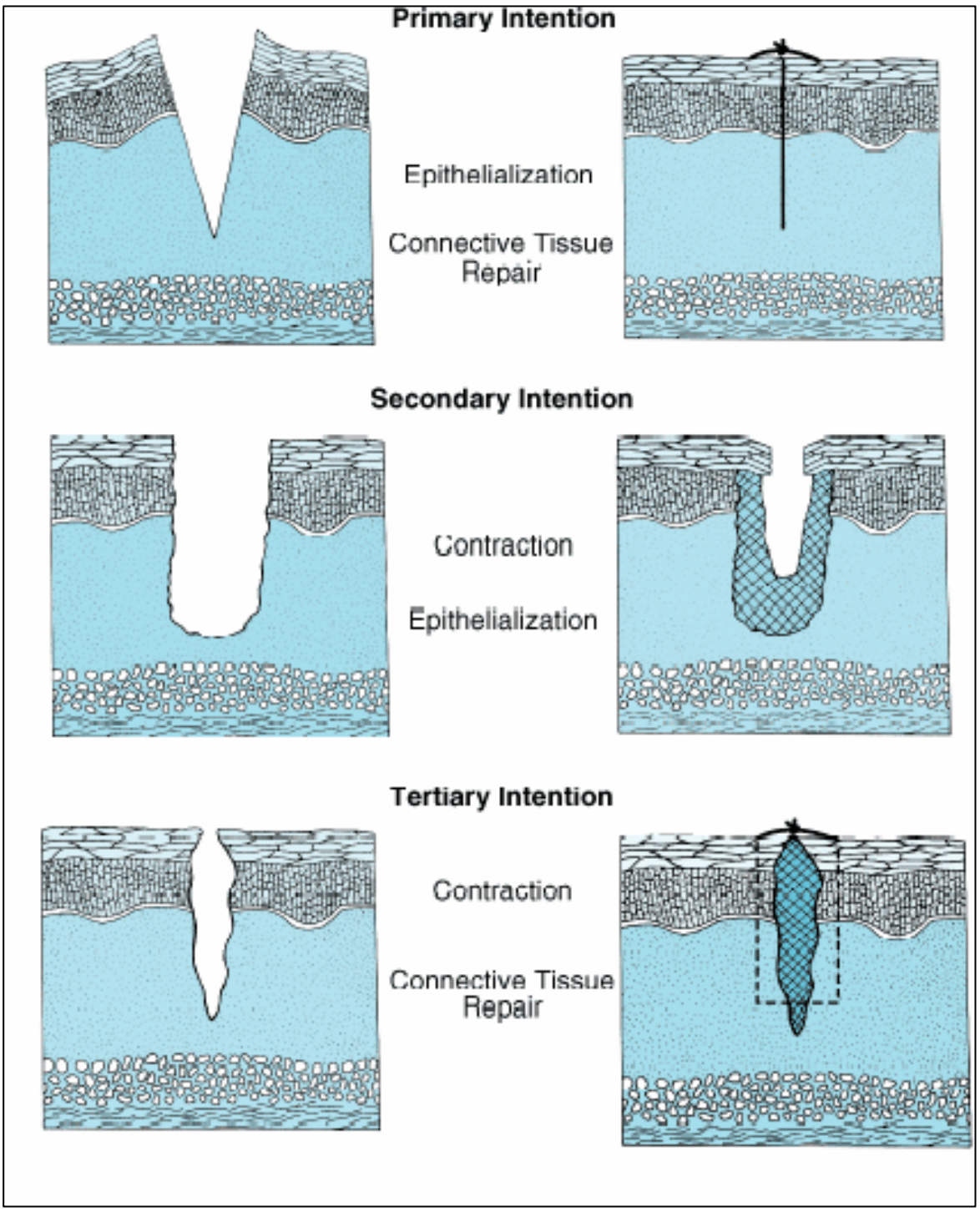


Figure 15 CLASSIFICATION OF WOUND HEALING

FACTORS THAT INHIBIT WOUND HEALING:[40]

1) **AGE -** the older the patient, the slower the healing.
2) **MALNUTRITION-** Through major direct and indirect effects, the normal healing mechanism is altered by malnutrition. In order to prevent infection, adequate nutrition is necessary which aids in maintaining a competent immune system. Normal tensile strength and collagen framework synthesis needs iron, magnesium, zinc, vitamins A and C, calories and adequate proteins. Delay in wound healing is due to protein catabolism.
3) **INFECTION -** Hypoalbuminemia predisposes to infection due to compromised immunity. Infected wounds lead to tissue destruction by prolonging the phases of inflammation, delaying collagen synthesis, preventing epithelialization and increasing the production of inflammatory mediators like cytokines. Presence of more than 10 bacteria signifies infection. Critical factors of infectivity are concentration, virulence, and host resistance.
4) **DIABETES MELLITUS (DM) -** Impaired wound healing in diabetic patients is well established. Reduced collagen synthesis and deposition and decreased tensile strength characterise the wound repair in patients with DM.

   A direct relationship between tensile strength and glycosylated haemoglobin levels have been described. The differences in wound repair are partially explained by increased levels of proteases, decreased levels of proliferative cytokines and abnormal insulin levels. DM also results in compromised perfusion; patients with DM are at high risk of microvascular diseases. Consequently, the delivery of micronutrients at the capillary level is impaired and there is an increase in vascular permeability. All these factors increase infection risk and diminished support for wound healing.

5) **OBESITY -** It is independently a risk factor for impaired wound healing. Adipose tissue is poorly vascularized. In addition, cardiac function is frequently compromised in obese patients, further diminishing tissue perfusion. As a result, infection, seroma formation, anastomotic leak and wound dehiscence are all more common among the obese population.

6) **MEDICATION -** Chemotherapeutic drugs and NSAIDS are the medications compromising wound repair. Former have an impact over rapidly dividing cells. The cells more profoundly affected are fibroblasts and myofibroblasts which would result in impaired collagen synthesis and wound contraction.
7) **STRESS** - It impairs wound healing as a potential co factor with its physiological and psychological means. Stimulation of sympathetic nervous system and elevation of serum corticosteroid levels leads to compromise of immune function and perfusion due to vasoconstriction as a result of stress.
8) **IMMUNOSUPPRESSION-** Immunosuppression increase susceptibility to infection and retards wound healing and this is attributed primarily to impairment of the inflammatory process.
9) **RADIATION THERAPY -** It causes damage to the keratinocytes and fibroblasts as well as nutrient blood vessels resulting in delayed healing.
10) **CIGARETTE SMOKING -** It affects both perfusion and oxygenation. Nicotine, carbon monoxide, and hydrogen cyanide are the three by-products of cigarette smoking. Nicotine is a potent vasoconstrictor and potentiates platelet aggregation, the oxygen saturation is lowered due to carbon monoxide and cellular oxygen transport is depleted by hydrogen cyanide. Therefore, there exists a high incidence of wound infection, dehiscence and delayed healing among smokers.[40]
11) **LOCAL FACTORS -** The local factors that affect the repair process are wound bed desiccation, pH, hypothermia, excess wound fluid, and/ or heavy bacterial colonization. A moist, clean wound surface that is maintained at a temperature of about 30 degrees Celsius supports wound healing to the best.

NUTRIENT	ROLE IN HEALING
Protein	Fibroplasia
	Angiogenesis
	Collagen formation and wound remodeling
	Immune function
	Precursor nitric oxide
Carbohydrates	Energy supply
	Protein sparing
	By-product of lactate
	Angiogenesis
Fat	Formation and stability of cell walls and intracellular organelles
	Inflammation
Vitamin A*	Epithelialization
	Wound closure
	Inflammatory response
	Angiogenesis
	Collagen formation
B vitamins	Cofactor in enzyme systems
	Immune response
	Synthesis of protein, fat, and carbohydrate
Vitamin C	Collagen synthesis
	Capillary wall integrity
	Fibroblast function
	Immunologic function
	Antioxidant
Vitamin D*	Calcium metabolism for building and maintaining bone
Vitamin E*	Unknown in relation to wound healing
	Antioxidant
Vitamin K*	Coagulation
Copper	Cross-linking of collagen
	Erythropoiesis
Iron	Collagen formation
	Leukocyte function
	Oxygen transport
Magnesium	Protein synthesis
Zinc	Collagen formation
	Protein synthesis
	Cell membrane stability
	Host defenses

*Fat-soluble vitamins.

Figure 16 THE ROLE OF NUTRIENTS IN WOUND HEALING

SURGICAL SITE INFECTION

These refer to the infections that are present along a surgical tract in any location after a surgical procedure. SSI is defined by the Centre for Disease Control and Prevention as the one that can occur on any post operative day ranging from 0 to 30 days to as late as 1 year in procedures such as implantation of mesh.[43]

Classification

1) Incisional superficial SSI
2) Incisional deep SSI
3) Organ/space related

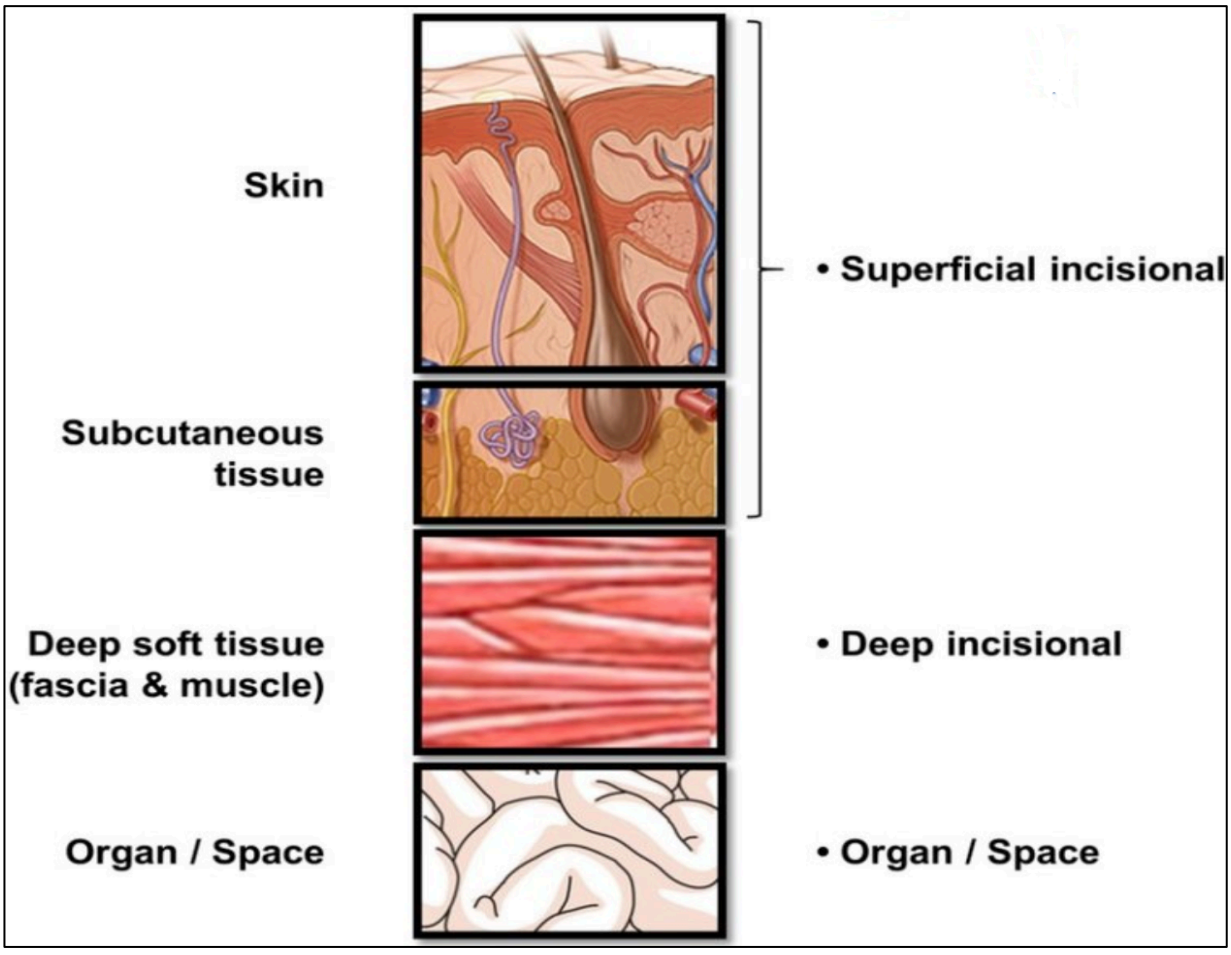


Figure 17 CLASSIFICATION OF SURGICAL SITE INFECTION

SUPERFICIAL INCISIONAL SSI

Infection that occurs within the 30th post operative day involving only the subcutaneous tissue and skin along with either one of the following

1) Purulent discharge
2) Isolating organisms from aseptically cultured tissue and fluid.
3) At least one sign of infection: Localised swelling, tenderness or pain, warmth and incision are deliberately opened by surgeon unless the incision is culture negative
4) Diagnosis of SSI by an attending physician or surgeon.[44]

Figure 18 SUPERFICIAL INCISIONAL SSI

DEEP INCISIONAL SSI

Infection occurring within 30 days of surgery or within 1 year of operation if implants are in place and infection involving deep soft tissue; and at least one of the following

1) Purulent discharge
2) Deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following symptoms; fever more than 38-degree Celsius, localized pain or tenderness unless the site is culture negative.
3) Evidence of deep infection on direct examination, during reoperation or on radiological examination.
4) Diagnosis of SSI by an attending physician or surgeon.[43,44]

Figure 19 DEEP INCISIONAL SSI

ORGAN/SPACE SSI

Infection occurring within 30 days of surgery or within 1 year of operation if implants are in place and infection involving any part of anatomy that was manipulated during an operation, other than the incision and at least one of the following:

1) Purulent discharge that is placed through a stab wound into the organ space.
2) Organisms isolated from an aseptically cultured fluid or tissue. Evidence of deep infection on direct examination, during reoperation, or on radiological examination.
3) Diagnosis of SSI by an attending physician or surgeon.

About 38% of all infections in surgical patients are due to nosocomial infections among which SSI are more common. Among them incisional SSI are the most common. Organ /space related SSI account for 93% of SSI related mortality and thus have worser prognosis than incisional SSI's that account for 60 - 80% of all SSI's.

The most common pathogen in SSI is Staphylococcus aureus followed by coagulase negative staphylococcus, enterococci, and E. coli. Bacterial contamination of >10 organisms frequently cause infection whereas contamination with < 10 organisms usually does not.[43,44]

Figure 20 PURULENT DISCHARGE FROM BURST ABDOMEN[45]

Synthetic Hernia Mesh

Mesh is either synthetic – a knitted material or non-knitted sheet. It may be absorbable, or non-absorbable or a combination of both. Non- absorbable mesh, which is mostly synthetic, will stay in the body indefinitely. It is a permanent implant and difficult to remove.

Animal-Derived Hernia Mesh Animal-derived mesh can be made from animal tissues, such as the intestine or skin of a pig (porcine) or cow (bovine) that is processed and sterilized. Animal-derived mesh is absorbable.

Over time, absorbable mesh will degrade and lose strength while new tissue growth takes over to strengthen the repair.[46]

Complications associated with hernia mesh are[47]

1) Mesh related adverse reactions
2) Adhesions
3) Injuries to adjacent vasculature, neuronal structures or organs
4) Additional complications with or without mesh can include infection, chronic pain and hernia recurrence.

Complications can include pain, nerve entrapment, reduced movement, and a foreign object reaction that can include fever, swelling, and general malaise.[48]

Figure 21 INFECTED HERNIA MESH

SURGICAL WOUND CLASSIFICATION ACCORDING TO DEGREE OF CONTAMIMATION

1) **Clean**

Uninfected operative wound in which no inflammation is encountered and respiratory, alimentary, genital, or infected urinary tract is not entered. Wounds are closed primarily and if necessary drained with closed drainage. Surgical wounds after blunt trauma should be included in this if they meet the criteria.[49]

2) **Clean contaminated**

An operative wound in which the respiratory, alimentary, genital or urinary tract is entered under controlled conditions and without unusual contamination.[49]

3) **Contaminated**

Open, fresh, accidental wounds. In addition, operation with major break in the sterile technique or gross spillage from gastrointestinal tract and incision in which acute non purulent inflammation is encountered are included in this category.[49]

4) **Dirty**

Old traumatic with retained and devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation. The accepted range of infection rates has been 1-5% for clean, 3%-11% for clean contaminated, 10%-17% for contaminated, and >27% for dirty wounds.[49]

Grade	Appearance
0	Normal healing
1	Normal healing with mild bruising or erythema
1a	Some bruising
1b	Considerable bruising
1c	Mild erythema
2	Erythema + other signs of inflammation
2a	At one point
2b	Around sutures
2c	Along wound
2d	Around wound
3	Clear or haemoserous discharge
3a	At one point only (<2cm)
3b	Along wound (>2cm)
3c	Large volume
3d	Prolonged (>3days)
Major complications	
4	Pus
4a	At one point only (<2cm)
4b	Along wound (>2cm)
5	Deep or serve wound infection with or without tissue breakdown; hematoma requiring aspiration

SOUTHAMPTON WOUND GRADING SYSTEM

Adogwa O et al showed that retrospective cohort study. To investigate the association between preoperative baseline serum albumin and postoperative surgical complication. The prevalence of malnutrition in the hospitalized patient population has only been recently recognized. Preoperative hypoalbuminemia (serum albumin <3.5 g/dL) has been shown to be associated with increased morbidity and mortality rates. The prognostic implications and significance of hypoalbuminemia after spine fusion surgery remain unknown. In this study, they assess the predictive value of preoperative nutritional status (serum albumin level) on postoperative complication rates. The medical records of 136 consecutive patients undergoing spine fusion at Duke University Medical Centre were reviewed. Preoperative serum albumin level was assessed on all patients and used to quantify nutritional status. Albumin less than 3.5 g/dL was recognized as hypoalbuminemia (malnourished). Patient demographics, comorbidities, and postoperative complication rates were collected. Patients were also stratified into 2 groups based on their cause, namely elective degenerative/deformity versus nonelective cases. The association between preoperative serum albumin level and postoperative complication was assessed via logistic regression analysis. Overall, 40 (29.4%) patients experience at least 1 postoperative complication. Patients undergoing elective surgery had more complications based on preoperative albumin levels (malnourished patients: 35.7% vs. nourished patients: 11.7%, P = 0.03), whereas those undergoing nonelective surgery had similar. The manuscript submitted does not contain information about medical device(s)/drug(s). The substantial prevalence of malnutrition in the hospitalized patient population has only recently become more widely recognized. As many as 40% of adult hospitalized patients are malnourished; and an estimated 4.3% of community-dwelling adults are undernourished. 1–4 Moreover, preoperative malnutrition has been associated with a number of poor postsurgical outcomes, including infection, acute kidney injury, and mortality. 5–13 Despite the growing body of literature on the effects of hypoalbuminemia on postoperative outcomes in other surgical fields, few studies to date have investigated preoperative hypoalbuminemia in patients undergoing spine surgery. 2,14,15 As such, they designed a retrospective cohort analysis to assess the impact of preoperative hypoalbuminemia on complication rates after spinal fusion surgery in 2 groups of patients who are stratified by risk: (1) patients undergoing elective surgery for degenerative and deformity causes and (2) patients undergoing surgery for higher-risk causes, including traumatic and neoplastic causes. The primary aim of this study was to assess whether poor preoperative nutritional status is associated with greater complication rates (malnourished patients: 46.5% vs. nourished

patients: 42.1%, P = 0.75). For patients undergoing elective spinal surgery, logistic regression with and without propensity score adjustment for risk factors, demonstrated that preoperative serum albumin level was a significant predictor of postoperative complications (OR: 4.21; 95% CI: 1.09–16.19; P = 0.04) and (OR: 4.54; 95% CI: 1.17–19.32; P = 0.04), respectively. Preoperative hypoalbuminemia is an independent risk factor for postoperative complications after elective spine surgery for degenerative and deformity causes, and should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical outcomes.[50]

Bhandari TR et al found that hypoalbuminemia, as a marker of malnutrition and disease, is associated with higher risk of poor postoperative outcomes. However, no study has been reported from their country until now. Their aim was to identify the relationship between preoperative hypoalbuminemia and the development of complications after major gastrointestinal surgery. A prospective study of 106 patients who underwent major elective gastrointestinal surgery between July 2012 to June 2013 in Department of Surgery, Tribhuvan University Teaching Hospital, Kathmandu, Nepal was considered for the study. Serum albumin was determined preoperatively in all patients. Hypoalbuminemia was defined as albumin less than 35 mg/dl. Thirty-day postoperative complications were analysed. The patients were 70 males (66 %) and 36 females (34%) with a mean age of 50 years (23-81). Thirty-six patients (34%) had hypoalbuminemia. Overall complication rate was 32%. Hypoalbuminemia patients had a significantly higher rate of postoperative complications (26.4 and 5.6%; P<0.05). In multivariate analysis, age (>50 year), BMI (<18 kg/m2), duration of surgery (>3 hours) and hypoalbuminemia (<35 mg/dl) were the significant risk factors for postoperative complications (P<0.05). Preoperative hypoalbuminemia is an independent predictor of postoperative outcomes in patients with elective major gastrointestinal surgery. Identification and optimization of nutritional status prior to surgery may improve surgical outcomes.[51]

Labgaa I et al found that to test postoperative serum albumin drop (ΔAlb) as a marker of surgical stress response and early predictor of clinical outcomes. Prospective cohort study (NCT02356484). Albumin was prospectively measured in 138 patients undergoing major abdominal surgery. Blood samples were collected before surgery and on postoperative days 0, 1 2 and 3. ΔAlb was compared to the modified estimation of physiologic ability and surgical stress (mE-PASS) score and correlated to the performances of C reactive protein (CRP), procalcitonin (PCT) and lactate (LCT). Postoperative outcomes were postoperative complications according to Clavien classification and Comprehensive Complication Index

(CCI), and length of hospital stay (LoS). Department of abdominal surgery in a European tertiary centre. Adult patients undergoing elective major abdominal surgery, with anticipated duration ≥2 hours. Patients on immunosuppressive or antibiotic treatments before surgery were excluded. The level of serum albumin rapidly dropped after surgery. ΔAlb correlated to the mE-PASS score (r=0.275, p=0.01) and to CRP increase (r=0.536, p<0.001). ΔAlb also correlated to overall complications (r=0.485, p<0.001), CCI (r=0.383, p<0.001) and LoS (r=0.468, p<0.001). A ΔAlb ≥10 g/L yielded a sensitivity of 77.1% and a specificity of 67.2% (AUC: 78.3%) to predict complications. Patients with ΔAlb ≥10 g/L on POD 1 showed a threefold increased risk of overall postoperative complications. Early postoperative decrease of serum albumin correlated with the extent of surgery, its metabolic response and with adverse outcomes such as complications and length of stay. A decreased concentration of serum albumin ≥10 g/L on POD 1 was associated with a threefold increased risk of overall postoperative complications and may thus be used to identify patients at risk.[52]

Chiang JM et al found that the quantitative relationship between serum albumin level and surgical outcomes has not been clearly established. This study included 3732 patients with colon cancer who underwent a potentially curative colectomy. Post-operative mortality and morbidity were analysed according to the patients' demographic data, pre-operative comorbidities, and tumour-related factors. Age, asthma, renal impairment, and albumin level were significantly associated with post-operative morbidity and mortality in the multivariate analyses. Logistic regression analysis revealed linear relationships of post-operative morbidity and mortality with albumin level. The morbidity and mortality rates decreased by 7.3% and 15.6%, respectively, for each 0.1 g/dL increase in albumin level. This finding remained significant in the hypalbuminaemia subgroup but not in the normoalbuminaemia subgroup. That is, the morbidity and mortality rates significantly decreased by 8.7% and 17.7%, respectively (both $P < 0.001$), in the former group and decreased by 2.7% ($P = 0.112$) and 11.6% ($P = 0.092$), respectively, in the latter group. This study demonstrated that serum albumin level linearly predicted the post-operative morbidity and mortality among the colorectal cancer patients. Pre-operative serum albumin level may therefore be used as a continuous rather than a categorical marker of disease severity, especially among patients with hypalbuminaemia.[53]

Inagaki E et al found that the effect of preoperative malnutrition on outcomes in patients undergoing major vascular surgery is unclear. They investigated the effects of

preoperative hypoalbuminemia, a marker for malnutrition, on outcomes after open abdominal aortic aneurysm repair (OAR) and endovascular abdominal aortic aneurysm repair (EVAR). Patients undergoing OAR or EVAR were identified in the 2005 to 2012 American College of Surgeons National Surgical Quality Improvement Program database and stratified into three groups: normal albumin (serum albumin >3.5 g/dL), moderate hypoalbuminemia (2.8-3.5 g/dL), and severe hypoalbuminemia (<2.8 g/dL). Multivariable analyses were performed to assess the association of preoperative hypoalbuminemia with 30-day morbidity and mortality. They identified 15,002 patients with a recorded preoperative serum albumin who underwent OAR (n ¼ 4956) or EVAR (n ¼ 10,046). Patients in both cohorts with hypoalbuminemia had a higher burden of comorbidity. In OAR patients, multivariable analyses demonstrated that moderate hypoalbuminemia was associated with an increased risk of 30-day mortality (odds ratio [OR], 1.32; 95% confidence interval [CI], 1.02-1.70) and postoperative length of stay (LOS; means ratio [MR], 1.10; 95% CI, 1.04-1.16), whereas severe hypoalbuminemia was associated with increased 30-day mortality (OR, 1.92; 95% CI, 1.37-2.70), reoperation #30 days (OR, 1.80; 95% CI, 1.32-2.48), pulmonary complications (OR, 1.40; 95% CI, 1.06-1.86), and postoperative LOS (MR, 1.33; 95% CI, 1.21-1.45). In EVAR patients, moderate hypoalbuminemia was associated with an increased risk of 30-day mortality (OR, 1.90; 95% CI, 1.38-2.62), pulmonary complications (OR, 1.61; 95% CI, 1.26-2.04), reoperation #30 days (OR, 1.39; 95% CI, 1.12-1.74), and postoperative LOS (MR, 1.23; 95% CI, 1.18-1.29), whereas severe hypoalbuminemia was associated with increased 30-day mortality (OR, 2.98; 95% CI, 1.96-4.53), pulmonary complications (OR, 1.88; 95% CI, 1.32-2.67), reoperation #30 days (OR, 1.54; 95% CI, 1.08-2.19), and postoperative LOS (MR, 1.52; 95% CI, 1.40-1.65). Preoperative hypoalbuminemia is associated with increased postoperative morbidity and mortality in a severity-dependent manner among patients undergoing OAR or EVAR. Evaluation and optimization of nutritional status should be performed preoperatively in this high-risk population.[54]

Sharma L et al showed that protein energy malnutrition affects every organ system. So correct assessment of nutritional status is important as malnourishment is a risk factor for morbidity and mortality in surgical patients. In their study, serum albumin has been taken as parameter for nutritional assessment. Study is conducted on 50 patients admitted in Department of General Surgery, Dr. Sampurnanand Medical College and associated Hospital, Jodhpur, Rajasthan, India for surgeries during January 2014-December 2015. Data was analysed using

Z-test and Fischer exact t-test and p value was calculated. P value <0.05 is considered statistically significant. Patients with serum albumin <3gm/dl had more complications with maximum number of complications in age group of 41–60-year group. This finding was statistically significant. Low serum albumin patients have more post-operative complication than those with normal level and so is a good indicator of post-operative morbidity and mortality.[55]

Li P et al found that a decrease in serum albumin is commonly observed after lung cancer surgery; however, whether it is associated with postoperative outcomes is unknown. The objective of this study was to evaluate whether the reduction of serum albumin (ΔALB) on postoperative day one could serve as a predictor of postoperative pulmonary complications (PPCs) after thoracoscopic anatomical resection in lung cancer patients. Patient's characteristics were compared between groups of whether they experienced PPCs or not. The cut-off value of ΔALB was examined by receiver operating characteristic curve to find out the threshold value of ΔALB in predicting PPCs. Logistic regression analysis was conducted to identify potential risk factors for PPCs. Totally 533 patients were included into analysis, and among them, 52 experienced PPCs. The ΔALB was significant in the PPCs group than in the non-PPCs group (P<0.001), and ΔALB was observed an independent risk factor for PPCs (OR =2.268, 95% CI: 1.153–4.460). The cut-off value of ΔALB in predicting PPCs was 14.97%. Patients with ΔALB ≥14.97% were more likely to have PPCs (P<0.001). A reduction of serum albumin with a cut-off value of 14.97% can be served as a predictor to identify patients at high risk of developing PPCs following thoracoscopic anatomical lung cancer surgery.[56]

Wang Y et al observed that femoral neck fracture usually occurs in the geriatric population. Postoperative pneumonia (POP) is known to be devastated, and it is the most frequent complication among patients receiving surgical treatment for femoral neck fractures. However, whether patients who have hypoalbuminemia are susceptible to the development of POP is a serious concern, although it has not been investigated. They attempted to investigate the association between newly developed POP and hypalbuminaemia and to identify whether hypoalbuminemia is an independent risk factor for POP after femoral neck fracture in geriatric population. They retrospectively reviewed the records from the first 30 days after surgery of patients who were ≥65 years of age and who had a femoral neck fracture treated with surgery between January 2018 and December 2018 at the Honghui Hospital, Xi'an Jiaotong University. Patients were divided into two groups based on whether they did or did not experience POP,

and their clinical characteristics were compared. Binomial logistic regression was used to identify potential risk factors of POP by analysing demographic factors, preoperative comorbidities, laboratory results, and surgical factors. A total of 720 patients were included in the analysis, and 54 patients experienced POP. The incidence of POP after surgical treatment for a femoral neck fracture in this geriatric population was 7.5%. In addition, patients with POP had significantly longer hospital stays than patients without POP. The binary logistic regression analysis revealed that preoperative hypoalbuminemia [odds ratio =5.187, 95% confidence interval (CI): 2.561–10.506, P<0.0001], COPD (OR =3.819, 95% CI: 1.247–11.701, P=0.019), prior stroke (OR =3.107, 95% CI: 1.470–6.568, P=0.003) and the time from injury to surgery (OR =1.076, 95% CI: 1.034–1.119, P<0.0001) were predominant and independent risk factors associated with POP after femoral neck fracture in this geriatric population. The current study revealed that among a geriatric population admitted for femoral neck fracture surgery, preoperative hypoalbuminemia was a predictor of POP, followed by COPD, prior stroke and the time from injury to surgery. Thus, patients who undergo femoral neck fracture surgery and have preoperative hypoalbuminemia should receive additional monitoring and perioperative care.[57]

Byakhodi KG et al showed that wound healing requires energy and is a catabolic process. Patients who are severely malnourished demonstrate impaired wound healing and predisposition to infection. They also suffer deficient immune mechanisms. The degree of malnutrition is estimated on the basis of weight loss during the past 6 months, physical findings and plasma protein assessment. The purpose of this study was to investigate serum albumin and BMI as predictors of postoperative mortality and morbidity. Total of 117 patients were included in the study admitted in KIMS Hubli from November 2012 to June 2014. Results and in this study serum albumin 3.5g/dl. But BMI did not show any statistically significance in the study.[58]

Kumar S et al hypoalbumenia has been shown to be associated with increased morbidity and mortality in acute surgical patients due to increased catabolism. This study intends to correlate between hypoalbumenia and postoperative complications in patients undergoing emergency abdominal surgery. A prospective study is done in a rural tertiary care centre in a total of 190 patients undergoing emergency abdominal surgery after obtaining ethical clearance. In this study preoperative serum albumin and postoperative complications including death up to postoperative day thirty were recorded and gathered using a checklist

designed for the study and analysed. Serum albumin less than 3.5g/dl is considered as hypoalbumenia in this study. The correlation between preoperative serum albumin and postoperative morbidity and mortality is assessed. In a total of 190 patients, 93 (48.9%) patients had morbidity and 27 (14.2%) patients had mortality. Preoperative serum albumin less than 3.5g/dl is found in 120 (63.1%) cases and 70 (36.9%) patients has same or more than 3.5g/dl. Patients with preoperative serum albumin less than 3.5g/dl has morbidity in 87 (45.8%) patients than that of normal preoperative serum albumin level which is 6 (3.1%) cases (p= <0.0001, chi-square =72.31). The total mortality is 27 (14.2%) in which all mortality is found in low albumin group compared to no mortality in patients with normal serum albumin. As the albumin level decreases the risk of morbidity and mortality increases with majority of complication in this study occurring in patients with albumin group less than 2.5g/dl to 3g/dl. Preoperative serum albumin is a good predictor of surgical outcome after emergency abdominal surgery.

Issangya CE et al showed that albumin is an important protein that transports hormones, fatty acids, and exogenous drugs; it also maintains plasma oncotic pressure. Albumin is considered a negative active phase protein because it decreases during injuries and sepsis. In spite of other factors predicting surgical outcomes, the effect of pre and postoperative serum albumin to surgical complications can be assessed by calculating the percentage decrease in albumin (delta albumin). This study aimed to explore perioperative serum albumin as a predictor of adverse outcomes in major abdominal surgeries. All eligible adult participants from Kilimanjaro Christian Medical Centre Surgical Department were enrolled in a convenient manner. Data were collected using a study questionnaire. Full Blood Count (FBP), serum albumin levels preoperatively and on postoperative day 1 were measured in accordance with Laboratory Standard Operating Procedures (SOP). Data was entered and analyzed using STATA version 14. Association and extent of decrease in albumin levels as a predictor of surgical site infection (SSI), delayed wound healing and death within 30 days of surgery was determined using ordinal logistic regression models. In determining the diagnostic accuracy, a Non-parametric Receiver Operating Curve (ROC) model was used. They adjusted for ASA classification, which had a negative confounding effect on the predictive power of the percent drop in albumin to adverse outcomes. Sixty-one participants were studied; the mean age was 51.6 (SD16.3), the majorities were males 40 (65.6%) and post-operative adverse outcomes were experienced by 28 (45.9%) participants. In preoperative serum albumin values, 40

(67.8%) had lower than 3.4 g/l while 51 (91%) had postoperative albumin values lower than 3.4 g/l. Only 15 (27.3%) had high delta albumin with the median percentage value of 14.77%. Delta albumin was an independent significant factor associated with adverse outcome (OR: 6.68; 95% CI: 1.59, 28.09); with a good predictive power and area under ROC curve (AUC) of 0.72 (95% CI 0.55 0.89). The best cutoff value was 11.61% with a sensitivity of 76.92% and specificity of 51.72%. Early perioperative decreases in serum albumin levels may be a good, simple and cost-effective tool to predict adverse outcomes in major abdominal surgeries.[59]

Tenny TC et al observed that albumin is a family of globular proteins, the most common of which are the serum albumins. All the proteins of the albumin family are water-soluble, moderately soluble in concentrated salt solutions, and experience heat denaturation. Albumins are commonly found in blood plasma and differ from other blood proteins in that they are not glycosylated. Substances containing albumins, such as egg white, are called albuminoids. It contributes to the oncotic pressure of plasma and to maintaining the distribution of extracellular fluid between the vascular and extravascular compartments. Hypoalbuminemia is associated with poor tissue healing, decreased collagen synthesis in the surgical wounds or at anastomotic sites, and also plays a role in the impairment of immune responses, such as macrophage activation and granuloma formation. Although it is well established that hypoalbuminemia, as a marker of malnutrition and disease, is associated with greater risk of adverse surgical outcome, previous studies have been based on relatively small samples and selected types of operations and have failed to adequately separate the predictive ability of albumin level from other risk factors. To determine whether preoperative serum albumin level can be used as an independent predictor of post-operative morbidity and mortality. This is a prospective comparison study conducted in general surgery department, Jubilee Mission Medical College. The source of the data was all patients who undergo gastrointestinal surgery with resection and anastomosis in the Department of General Surgery, Jubilee Mission Medical College, Thrissur from December 2017 to May 2019. Patients are investigated preoperatively for liver function test, renal function test, thyroid function test and urine- protein creatinine ratio (obtained from urine microscopy) to rule out secondary causes of hypoalbuminemia and are classified into two groups: - normal albumin level and hypoalbuminemia. Patients with normal serum albumin level are compared with patients of low serum albumin for length of hospital stay, resumption of oral diet, surgical site infection and mortality. The preoperative albumin levels ranged from 2.1 to 3.4 g/dl with a mean value of 3.03 g/dl. In most other similar

studies [1-4], a similar cut off normal range (3 or 3.5 g/dl) was assigned and patients were grouped into two based on this. To explore the possibility of using serum albumin as a simple and low-cost prognostic tool to predict the risk of adverse surgical outcomes, they used different statistical methods. Mann-Whitney U test has been performed to find out the significant role of albumin levels for length of hospital stay (Median 10.5 days and 8 days) and delay in resumption of diet (Median 5days and 3days) among two groups and it showed that there was statistically significant association with albumin levels between two groups in length of stay ($p=0.021$) if they classify the length of hospital stay as less than 10days and more than 10days. It is statistically significant in resumption of diet ($p<0.001$). There was a significant relationship found between the occurrence of surgical site infection and albumin levels. Where it was also found that those with low serum albumin had increased rates of complications (60%) against those with normal serum albumin levels 32.5%.15% mortality cases were reported; of which 22.5% falls in low serum albumin level group where as 7.5% in normal albumin level group. In view of these results, they conclude that preoperative albumin is a good prognostic indicator for predicting the outcome of surgery. It is a cheap and easily applicable test. By estimating albumin levels in surgical patients before surgery and adequately correcting lower levels where necessary, post-operative morbidity could be reduced in these patients.[60]

Lalhruaizela S et al observed that the purpose of nutritional support in the surgical patient is to prevent or reverse the catabolic effects of disease or injury. Protein depletion results in delayed wound healing and many other complications. Serum albumin levels at the time of admission were found to be a very useful predictor of such complications. The ultimate validation for nutritional support in surgical patients should be improvement in clinical outcome and restoration of function. To find the relationship between serum albumin level <3 g/dL and the development of complications and mortality rate among patients following laparotomy for Gastrointestinal (GI) diseases. Ninety-nine patients, more than 18 years of age with serum albumin level <3 g/dL, posted for emergency and elective laparotomy for GI diseases were included in the study. The following assessments were done: pre-operative evaluation of serum albumin, nutritional assessment (Mid Upper Arm Circumference, Skin Fold Thickness) along with Body Mass Index (BMI) and post-operative evaluation of serum albumin after 48 hours and 7 days. Early and late (30 day) post-operative complications were studied. Statistical analysis was done by using descriptive and inferential statistics using Chi-square test and Multiple Regression Analysis. Maximum numbers of patients were in the age

group 40-59 years and 59 patients (59.60%) developed post-operative complications. Surgical Site Infection (SSI) was seen in majority (32.32%) of the patients as early post-operative complication and wound dehiscence was the most common late complication (9.09%). Most of the early post-operative complications were present in patients with serum albumin level 2.1-2.7 gm/dL (43.43%). The relation between pre-operative serum albumin and early and late post-operative complications was found to be statistically significant. There was a significant co-relation between age, BMI and complications. There were four deaths in the study. Pre-operative Hypoalbuminemia <3.0 gm/dL is a strong and independent risk factor for post-operative morbidity and mortality in GI surgeries and BMI is a strong indicator for post-operative complications.

Rathod SD et al found that albumin has crucial and markable functions in health. Hypoalbuminemia has been shown to be deeply associated with increased mortality and morbidity in hospitalized patients. Many studies were conducted in the past but none of them has a definitive result. With this literature review they aim to review near-past studies and find out the importance of preoperative serum albumin and its outcome on postoperative results.[61]

Reis TG et al found that malnutrition is a common issue in patients with head and neck squamous cell carcinoma and has a negative effect on surgical outcomes. They attempted to determine which malnutrition diagnostic variables can be used as predictors of postoperative complications in patients with head and neck squamous cell carcinoma. Forty-one patients undergoing surgery for head and neck squamous cell carcinoma were submitted to a prospective evaluation. Biochemical data, anthropometric measurements and evaluation of body composition were used in the nutritional analysis. Twenty-two patients (53.6%) developed complications. Serum albumin measured on the first postoperative day was the only variable that significantly differed between groups. A cut-off value of 2.8 g/dL distinguished between patients with a complicated and uncomplicated postoperative course. Normalization of albumin levels occurred more frequently and more rapidly in the noncomplicated group. Serum albumin measured on the first postoperative day was the only variable that was a predicter of postoperative complications after major head and neck squamous cell carcinoma surgery.[62]

Rathod SD et al showed that serum albumin has been considered as a very important factor in determining the clinical outcome of a patient undergoing major surgery. Patients with

low serum albumin pre-operatively have been encountered with poor outcome postoperatively. This study was conducted to assess the association between serum albumin concentration and postoperative outcome (morbidity / mortality). This prospective observational study was carried out in JNMC and AVBRH, Sawangi, Wardha, from September 2018 to September 2020. All patients undergoing emergency / elective major surgeries were included in the study. Detailed history of patients was recorded including chief complaints and any other previous medical history. 104 patients were included in the study of which maximum numbers of patients were seen in the age range of 35-64 years. The mean age being 48.19 [+ or -] 15.60 years. The gender distribution with more male predominance with 59.62 % participants being males and 40.38 % participants being females. Total 68 patients had postoperative complications, 40 being males and 28 being females. Highest numbers of complications in this study were seen in age group 55-64 years. 50 out of 73 patients with benign diseases developed complications and 18 out of 31 patients with malignant diseases developed complications. Serum albumin has become an important modality to define the clinical outcome of a patient. Surgeons have started using it as a marker to define the post-operative outcome of a patient undergoing surgery. Exogenous albumin administration has also showed promising results in improving the outcome of a patient in the post- operative period.[61]

Liu Q et al found that the albumin, a negative acute-phase protein, is important for perioperative morbidity, even in patients with normal preoperative levels. This study intends to determine the perioperative factors related with the postoperative reduction in serum albumin (ΔALB) and its influence on perioperative outcome in a paediatric general surgical cohort. This single-centre retrospective review included 939 paediatric patients who underwent major gastroenterology surgery from August 2010 to August 2019. The patients were dichotomized into a high ΔALB group (≥14.6%) and a low ΔALB group (<14.6%) based on the mean value of ΔALB (14.6%). the independent risk factors for ΔALB, were explored using the propensity score matching to minimize potential selection bias and subjected to method multivariable logistic regression model. Furthermore, in 366 matched patients, the influences of operating time on perioperative outcomes were analysed. Among the 996 patients reviewed, 939 patient records were enrolled in the final analysis. Controlling for other factors, multivariable analysis showed that a high CRP on POD 3 or 4 [odds ratio (OR) = 2.36 (95% CI, 1.51–3.86); p = 0.007], a longer operating time [OR = 1.18 (95% CI, 1.00–1.53); p = 0.014), and the presence of Charcot's triad [OR = 1.73 (95% CI, 1.05–2.83); p = 0.031] were factors

that predicted a high ΔALB level. A high ΔALB level was also related with gastrointestinal functional recovery delay, reflected by the postoperative defecation (p = 0.013) and bowel movement (p = 0.019) delay and the high occurrence of postoperative complications (16.1 vs. 10.9%, OR, 1.57; 95% CI, 1.02–2.41, P = 0.0026). The high ΔALB level was correlated with postoperative outcome. To obtain a safe recovery and discharge after a major abdominal operation, the above risk factors for ΔALB could be addressed in the perioperative period.[63]

Mozumder MN et al serum albumin has a close correlation with degree of malnutrition which is associated with poor outcome and quality of life after cardiac surgery. Hypoalbuminemia is associated with increased wound infection; prolonged hospital stays and death after major surgery. Hence, preoperative serum albumin level can be utilized to upgrade risk models which will further benefit the cardiac surgical patients without extra financial burden. Objective of this study was to evaluate the role of serum albumin as a predictor of outcome after valve replacement surgery. This comparative cross-sectional study was carried out at the department of cardiac surgery in BSMMU. The study population was 50, with two groups having 25 patients each. Grouping of patients were done with respect to a present cut off value for serum albumin. The period of study was from August, 2018 to February, 2020 and purposive sampling method was applied for this study. Data was collected by using a standardized semi-structured questionnaire and face to face interview. By demographic characteristics, mean age was significantly higher in group B (49.96 ± 8.69 years) than in group A (41.60 ± 11.16 years) (p=0.005). Mean BMI was lower in group B (20.88 ± 3.71 kg/m2) than in group A (22.26 ± 1.67 kg/m2), which was found statistically significant (p=0.006). In terms of postoperative outcome, total chest drain collection was significantly higher in group B (968.80 ± 183.49 ml) than in group A (816.00 ± 113.40 ml), (p=0.001). Duration of ICU stay were significantly longer in group B (4.60 ± 0.76 days) than in group A (3.92 ± 0.86 days) (p=0.005). Similarly, duration of hospital stay was significantly longer in group B (9.88 ± 1.56 days) than in group A (8.64 ± 0.81 days) (p=0.001). Overall morbidity was significantly higher in group B (48%) than in group A (20%) (p<0.05). Mortality rate was higher in group B (12%) than in group A (4%), but that was not found statistically significant (p>0.05). Pearson co-efficient correlation test showed strong inverse relationship of serum albumin with total chest drain, ICU stay and hospital stay following valve replacement surgery (r=-0.473, r=-0.448 & r=-0.487 respectively), which was most significant than age and BMI (p ≤ 0.001). Multivariate logistic regression analysis was done to assess the predictive value of serum albumin level, age

and BMI, where preoperative serum albumin level was found to be the most valuable predictor of postoperative outcome after valve replacement surgery (B=-2.251, OR 0.105, 95% CI 0.011-0.986, $p<0.05$). This study demonstrated that preoperative low serum albumin level is associated with increased morbidity and mortality after valve replacement surgery. Hence, preoperative serum albumin level can be used as a reliable predictor of postoperative outcome following valve replacement surgery.[64]

Larsen PB et al showed that major abdominal surgery in older and frail patients is associated with considerable morbidity and mortality. Plasma albumin is routinely measured in the clinic and has been proposed as an indicator of frailty. This study aimed to investigate if plasma albumin is a predictor of mortality in older patients undergoing open abdominal surgery. They conducted a single-centre, register-based retrospective study of patients, aged ⩾60 years who underwent one of 81 open abdominal surgical procedures. Patients operated on during the period from January 1st, 2000 to May 31st, 2013 were consecutively identified in the Danish National Patient Registry. Plasma albumin was measured within 30 days prior to surgery and the primary endpoint was 30-day postoperative mortality. 3,639 patients were included of whom 68.2% underwent emergency surgery. The rate of severe hypoalbuminemia (plasma albumin < 28 g/L) was 43.4%. Preoperative plasma albumin was lower in patients with a fatal 30-day outcome (mean 20.6 g/L vs 30.1 g/L in survivors, $p < 0.0001$). Other independent predictive parameters of 30-day mortality were age, male sex, and emergency surgery. They present an algorithm including these four variables for the prediction of 30-day mortality for patients aged ⩾60 years undergoing open abdominal surgery. Preoperative plasma albumin is a predictor of 30-day mortality in patients above 60 years of age following open abdominal surgery. Assessment of plasma albumin in conjunction with other risk factors such as age, sex, and surgical priority may improve preoperative decision-making.[65]

Babu CS et al Hippocrates recognized the side effects of malnutrition on the morbidity and mortality of patients. The most clinically useful parameter is serum albumin. It predicts perioperative morbidity and mortality. The main objective was to study the preoperative serum albumin and Body Mass Index as predictors of post-operative morbidity and mortality in elective major general surgeries. The study was conducted on 50 patients who underwent elective surgery at Calcutta National Medical College and Hospital, Kolkata between March 2013-Feb 2014. Z- Test and Fischer exact t-test were used for statistical analysis. Preoperative and post-operative parameters were taken. Preoperative parameters serum albumin level and

body mass index. Post-operative parameters- morbidity and mortality. Permission from institutional ethics committee was obtained. Out of 50 patients studied 18 (36%) were malignant and 32 (64%) were non-malignant. 20 patients developed complications, 4 (20%) were malignant and 16 (80%) were non-malignant. Among the patients with malignancy (18) only 4 had complications, of which 2 (11%) were <3.0 g/dl and 2 (11%) between 3.1- 3.5 g/dl. Albumin less than 2.5 g/dl ($p<0.001$) and BMI less than 20 Kg/m2 ($p<0.005$) and greater than 30 Kg/m2 ($p <0.005$) was associated with increase in post-operative complications. The correlation between the serum albumin and complication rate was statistically significant in the malignant diseases when considered separately. Serum albumin < 3.0 g/dl was associated with increased post-operative morbidity and mortality.

Silahli M et al examined that this study was to evaluate the mortality and morbidity of infants <1 year of age with intestinal obstruction requiring surgical intervention and to investigate the factors affecting mortality and hospital length of stay in paediatric surgery, including albumin-haemoglobin index. The records of gastrointestinal paediatric surgeries in the past 10 years of patients who were <1-year-old at Baskent University Konya Hospital were obtained from the hospital and retrospectively studied. Patient characteristics, especially the relationship between albumin haemoglobin index (AHI) and hospital duration and mortality, were examined. According to the surgical areas, it also subjected this relationship to further analyse in subgroups. There were 144 cases who fulfilled the inclusion criteria. Pre-operative serum AHI was analysed using receiver operating characteristics (ROC) curve analyzes. In the ROC analysis, AHI had a diagnostic value in predicting case discharge rates (area under the curve: 0.755, P = 0.001). When the cut-off point was set at 46.18, the sensitivity of the test was 57.5% and the sensitivity for predicting survival was 84%. In the logistic regression model to estimate survival, the odds ratio of AHI was 1.063 (confidence interval: 1.020–1.108, P = 0.004). In subgroup analyzes, AHI positively predicted survival in the NEC group and in the other group. In a linear regression model analysing the effect of AHI on hospital stay of length, AHI explained 10% of the variance in the hospital stay of length variable and significantly and negatively influenced the hospital length variable ($\beta = -0.319$, P = 0.05). In the linear regression model for subgroup analyzes, AHI significantly and negatively predicted hospital length of stay in the NEC and pyloric surgery groups, but positively predicted hospital length of stay in the perforation group. The AHI can be used as a valuable marker to predict the likelihood of discharge and length of hospital stay in paediatric surgical cases <1-year-old.[66]

MATERIALS AND METHODS

MATERIAL AND METHODS

Place of study - Department of General Surgery, Santosh Medical College & Hospital Ghaziabad

Duration of study - July 2021 to June 2022

Study design - Prospective observational study.

Sample size – 200 Patients

Study Technique - Convenient Sampling

Inclusion Criteria –

1) Males and females between age of 18 to 80 years
2) Major general surgeries (Laparotomy, Laparoscopic/ Open Cholecystectomy, Laparoscopic/ Open Inguinal or Ventral hernia repair, CA Breast, LSCS and any major abdominal surgical procedure).

Exclusion Criteria –

1) Patients' refusal to be a part of study.
2) Patients < 18 years and > 80 years of age.
3) Immunocompromised patients.
4) Cases of trauma.

Methodology - After taking the required consent, patients will be assessed under the following-

- Preoperative parameter: Serum albumin level
- Postoperative parameter

Morbidity

1) Wound infection and discharge
2) Wound dehiscence
3) Anastomotic leak
4) Burst abdomen
5) Delayed wound healing
6) Complications of scar – Keloid/ Hypertrophic Scar
7) Ventral hernia

Mortality - Death

Study Tools

1) History
2) Clinical examination (Preoperative, postoperative)
3) Anthropometry
4) Investigations

   Preoperatively

   - Routine
     1. Complete blood count
     2. Liver function tests including S. Albumin
     3. Fasting and post prandial blood sugar
     4. Serum urea, creatinine
   - Selective
     1. Ultrasonography of abdomen
     2. Chest X-ray
     3. Abdominal radiograph

   Postoperatively

   - Complete blood count
   - Liver function tests including S. Albumin
   - Fasting and postprandial blood sugar
   - Serum urea, creatinine
   - Chest X-ray
   - Histopathological report of resected specimen (if any)
   - Pus if found for culture and sensitivity

5) Operative notes
6) Follow-up – At 1st,2nd and 4th weeks.

Data management and Statistical Analysis

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analysed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables. Two-sample t-tests for a difference in mean involved independent samples or unpaired samples. Paired t-tests were a form of blocking and had greater power than unpaired tests. A chi-squared test ($\chi 2$ test) was any statistical hypothesis test wherein the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true. Without other qualification, 'chi-squared test' often is used as short for Pearson's chi-squared test. Unpaired proportions were compared by Chi-square test or Fischer's exact test, as appropriate.

Explicit expressions that can be used to carry out various t-tests are given below. In each case, the formula for a test statistic that either exactly follows or closely approximates a t-distribution under the null hypothesis is given. Also, the appropriate degrees of freedom are given in each case. Each of these statistics can be used to carry out either a one-tailed test or a two-tailed test.

Once a t value is determined, a p-value can be found using a table of values from Student's t-distribution. If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis.

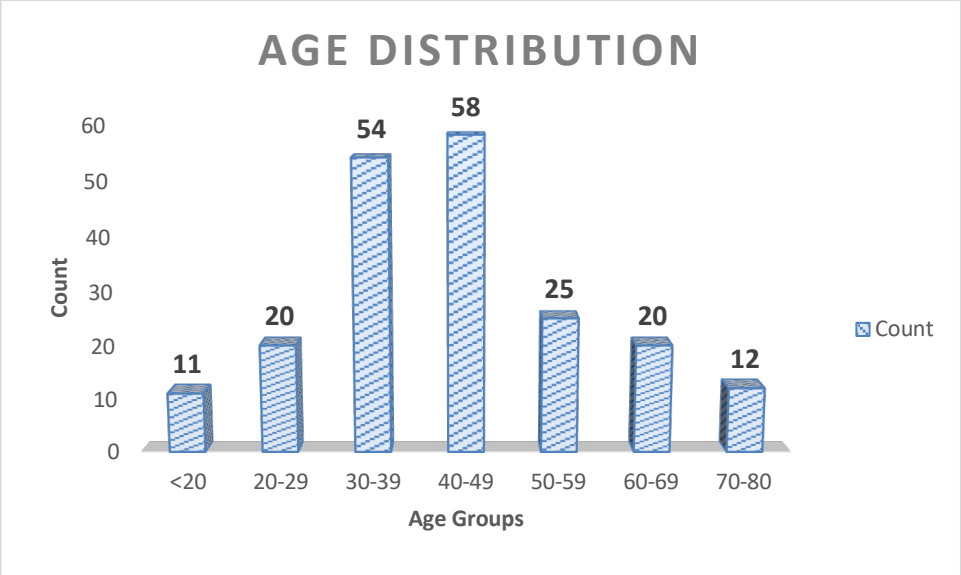
p-value ≤ 0.05 was considered for statistically significant.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

Table 1 AGE DISTRIBUTION

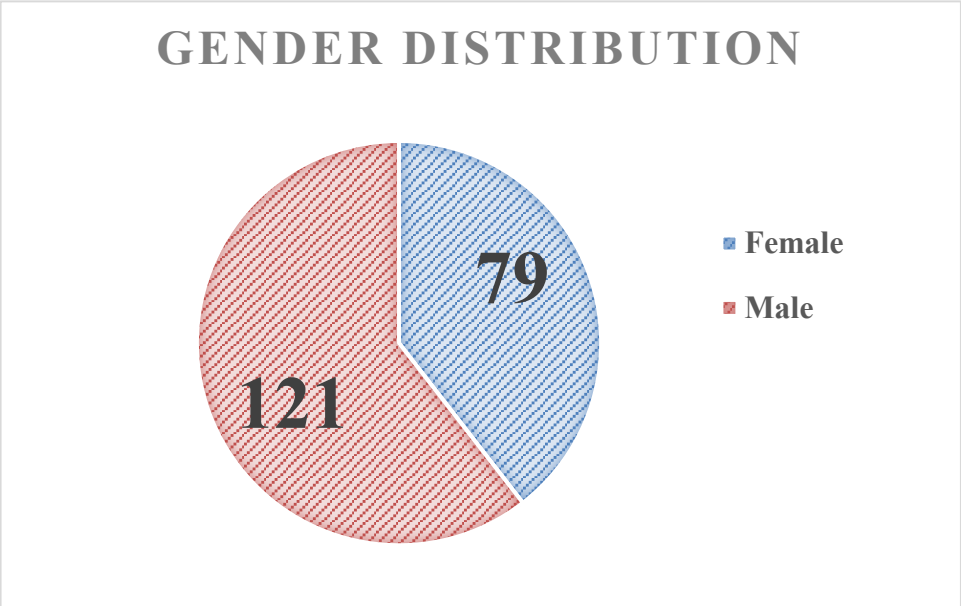
Age Groups	Count	Percentage
<20	11	5.50%
20-29	20	10.00%
30-39	54	27.00%
40-49	58	29.00%
50-59	25	12.50%
60-69	20	10.00%
70-80	12	6.00%
Grand Total	**200**	**100.00%**



In this study, 11 patients were <20 years of age, 20 patients were 20-29 years of age, 54 patients were 30-39 years of age, 58 patients were 40-49 years of age, 25 patients were 50-59 years of age, 20 patients were 60-69 years of age and 12 patients were 70-80 years of age.

Table 2 GENDER DISTRIBUTION

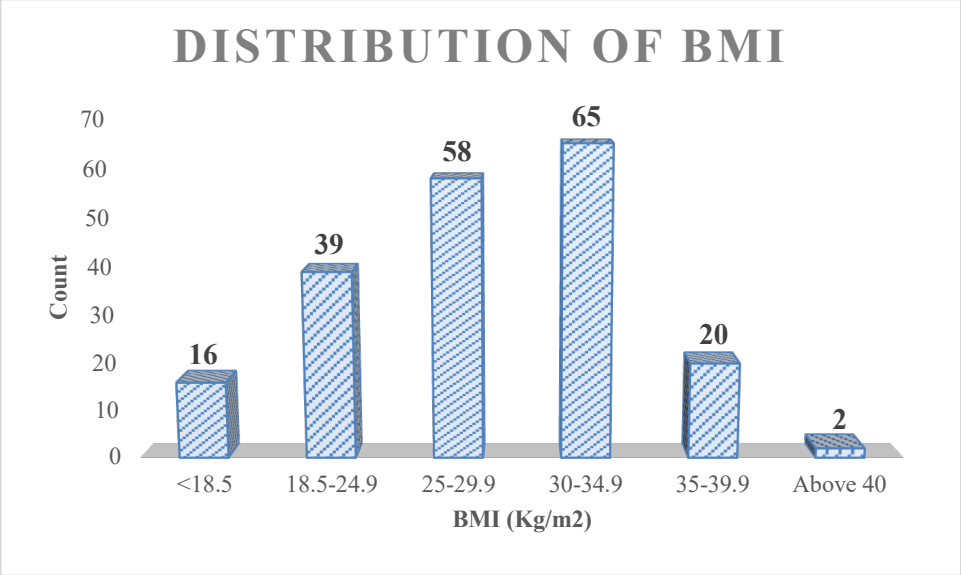
Gender	Count	Percentage
Female	79	39.50%
Male	121	60.50%
Grand Total	**200**	**100.00%**



Out of the 200 patients included in this study, 121 patients were male (60.50%) and 79 patients were females (39.50%).

Table 3 DISTRIBUTION OF BMI

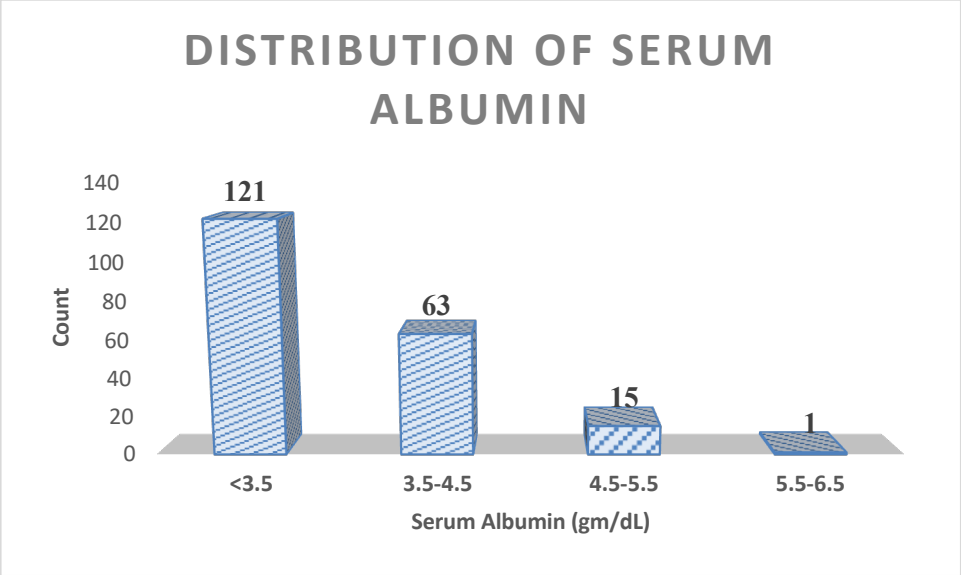
BMI Range (Kg/m2)	Count	Percentage
<18.5	16	8.00%
18.5-24.9	39	19.50%
25-29.9	58	29.00%
30-34.9	65	32.50%
35-39.9	20	10.00%
Above 40	2	1.00%
Grand Total	**200**	**100.00%**



In this study, 16 patients had <18.5 BMI, 39 patients had 18.5-24.9 BMI, 58 patients had 25-29.9 BMI, 65 patients had 30-35.9 BMI, 20 patients had 35-39.9 BMI and 2 patients had above 40 BMI.

Table 4 DISTRIBUTION OF SERUM ALBUMIN

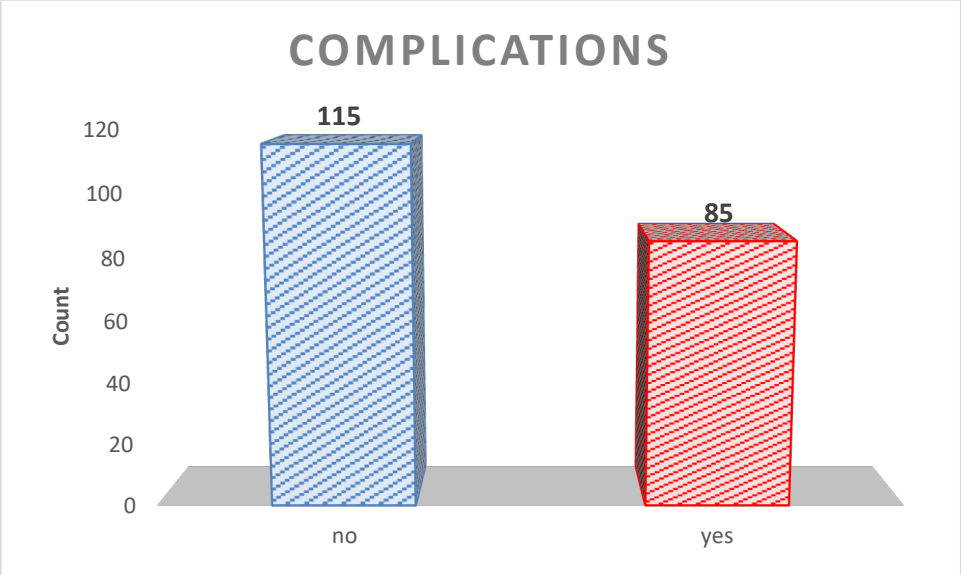
Serum Albumin (gm/dl)	Count	Percentage
<3.5	121	60.50%
3.5-4.5	63	31.50%
4.5-5.5	15	7.50%
5.5-6.5	1	0.50%
Grand Total	**200**	**100.00%**



In this study, 121 patients had <3.5 gm/dL S. Albumin, 63 patients had 3.5-4.5 gm/dL S. Albumin, 15 patients had 4.5-5.5 gm/dL S. Albumin and 1 patient had 5.5-6.5 gm/dL S. Albumin.

Table 5 POST-OPERATIVE OUTCOME

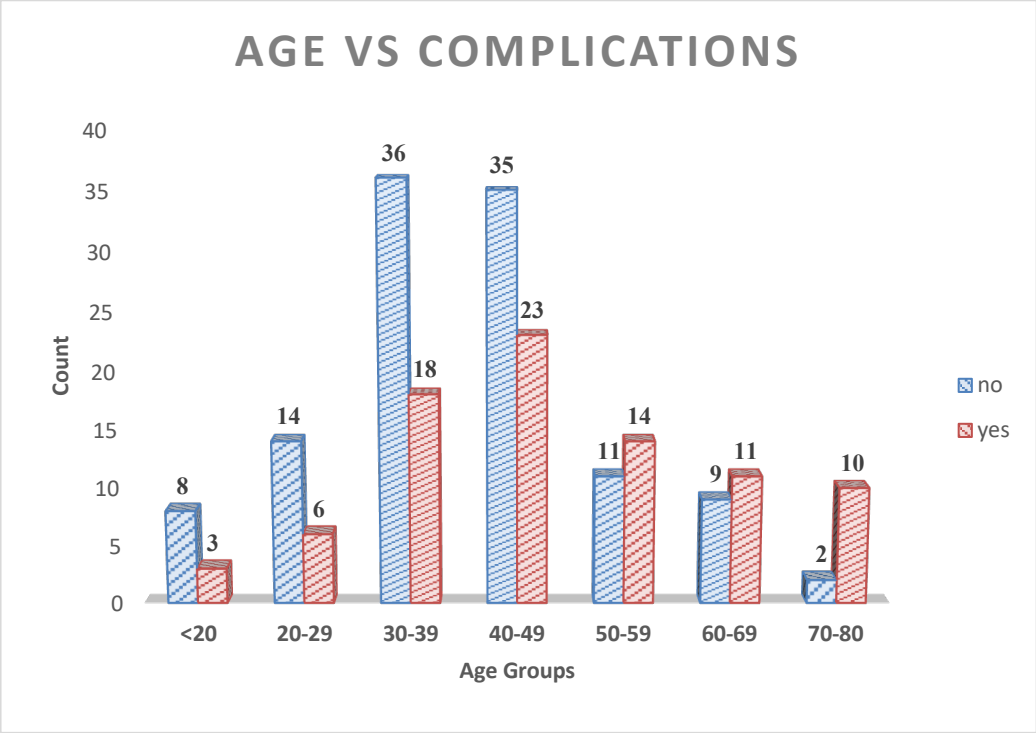
Complications	Count
No	115
Yes	85
Grand Total	**200**



Out of the 200 patients, 115 patients had no post-operative complications and 85 patients had post-operative complications.

Table 6 AGE VERSUS COMPLICATIONS

Age Groups	complications		
	No	Yes	Grand Total
<20	8	3	11
20-29	14	6	20
30-39	36	18	54
40-49	35	23	58
50-59	11	14	25
60-69	9	11	20
70-80	2	10	12
Grand Total	**115**	**85**	**200**



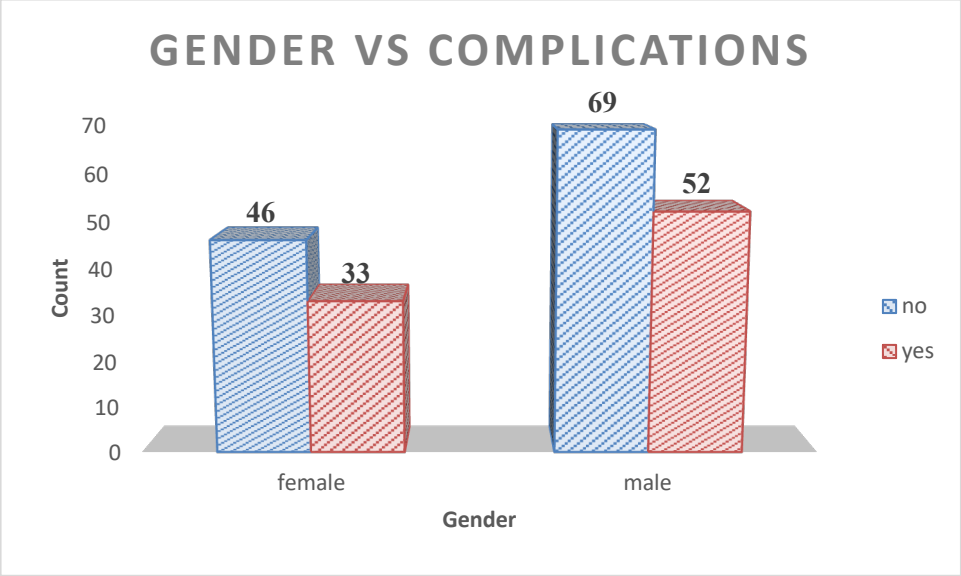
In without Complications, 8 patients were <20 years of age, 14 patients were 20-29 years of age, 36 patients were 30-39 years of age, 35 patients were 40-49 years of age, 11 patients were 50-59 years of age, 9 patients were 60-69 years of age and 2 patients were 70-80 years of age.

In with Complications, 3 patients were <20 years of age, 6 patients were 20-29 years of age, 18 patients were 30-39 years of age, 23 patients were 40-49 years of age, 14 patients were 50-59 years of age, 11 patients were 60-69 years of age and 10 patients were 70-80 years of age.

Association of Age with Complications was statistically significant (p=0.0154).

Table 7 GENDER VERSUS COMPLICATIONS

Gender	Complications		
	No	Yes	Grand Total
Female	46	33	79
Male	69	52	121
Grand Total	**115**	**85**	**200**

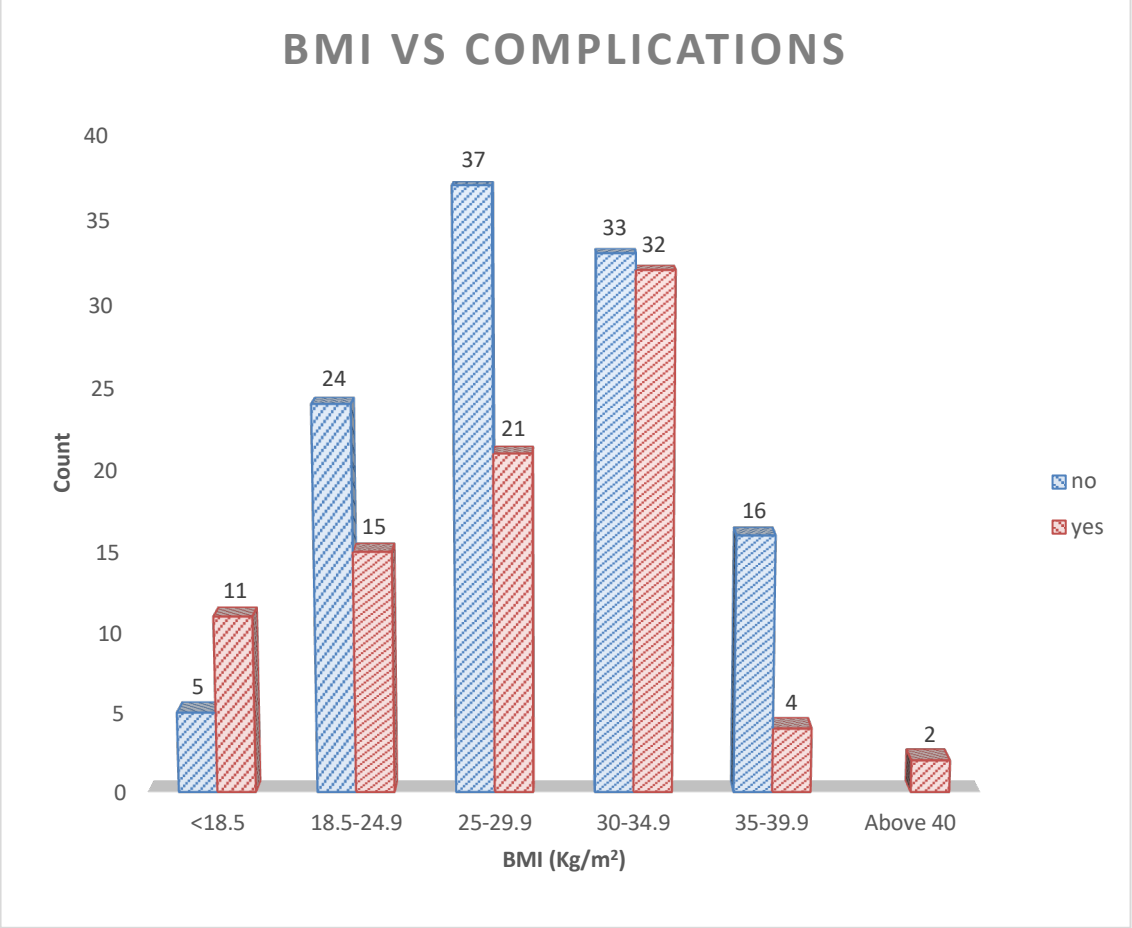


In without Complications, 46 patients were female and 69 patients were male. In with Complications, 33 patients were female and 52 patients were male.

Association of Gender with Complications was not statistically significant (p=0.5996).

Table 8 BMI VERSUS COMPLICATIONS

BMI (Kg/m2)	Complications		
	No	Yes	Grand Total
<18.5	5	11	16
18.5-24.9	24	15	39
25-29.9	37	21	58
30-34.9	33	32	65
35-39.9	16	4	20
Above 40		2	2
Grand Total	**115**	**85**	**200**



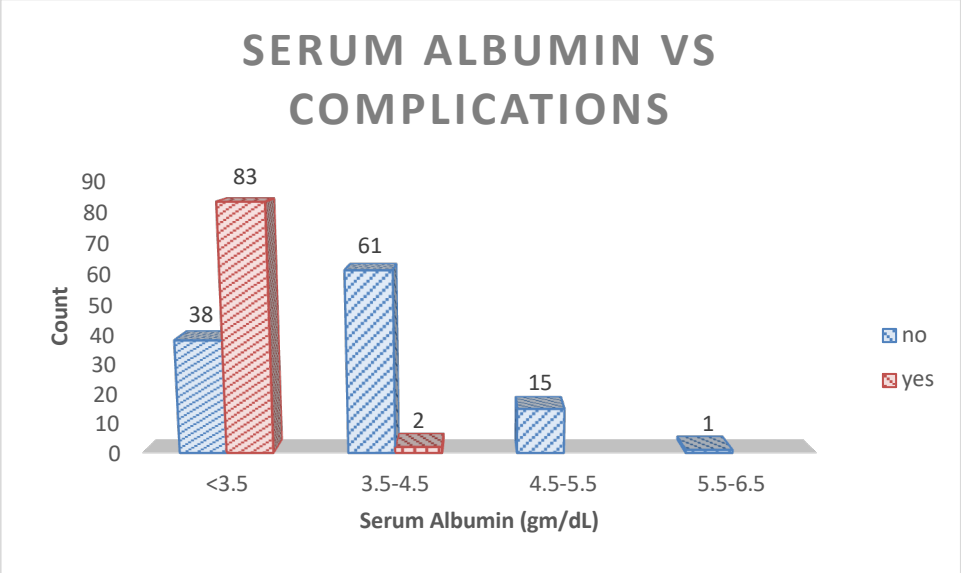
In without Complications, 5 patients had <18.5 BMI, 24 patients had 18.5-24.9 BMI, 37 patients had 25-29.9 BMI, 33 patients had 30-34.9 BMI and 16 patients had 35-39.9 BMI.

In with Complications, 11 patients had <18.5 BMI, 15 patients had 18.5-24.9 BMI, 21 patients had 25-29.9 BMI, 32 patients had 30-34.9 BMI, 4 patients had 35-39.9 BMI and 2 patients had above 40 BMI.

The mean difference of BMI (Kg/m^2) - Complications was 26.9825 with 95% confidence interval [26.1892 to 27.7758]. The difference of mean BMI (Kg/m^2) - Complications was statistically significant ($p<.001$).

Table 9 SERUM ALBUMIN VERSUS COMPLICATIONS

Serum Albumin	Complications		
	No	Yes	Grand Total
<3.5	38	83	121
3.5-4.5	61	2	63
4.5-5.5	15	0	15
5.5-6.5	1	0	1
Grand Total	**115**	**85**	**200**



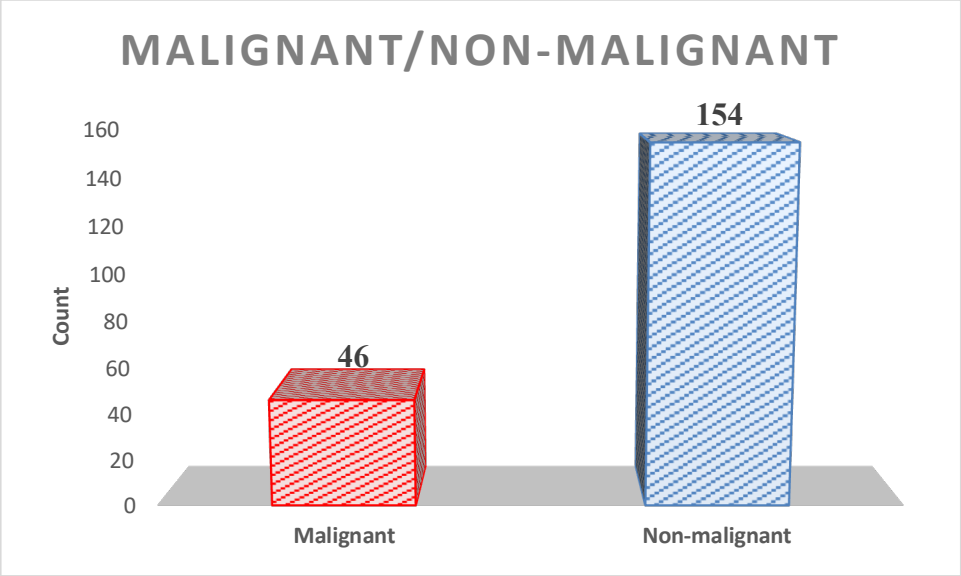
In without Complications, 38 patients had <3.5 S. Albumin, 61 patients had 3.5-4.5 S. Albumin, 15 patients had 4.5-5.5 S. Albumin and 1 patient had 5.5-6.5 S. Albumin.

In with Complications, 83 patients had <3.5 S. Albumin and 2 patients had 3.5-4.5 S. Albumin.

The mean difference of Serum Albumin (gm/dL) - Complications was 2.98955 with 95% confidence interval [2.83006 to 3.14904]. The difference of mean BMI (Kg/m2) - Complications was statistically significant (p<.001).

Table 10 MALIGNANT VERSUS NON-MALIGNANT

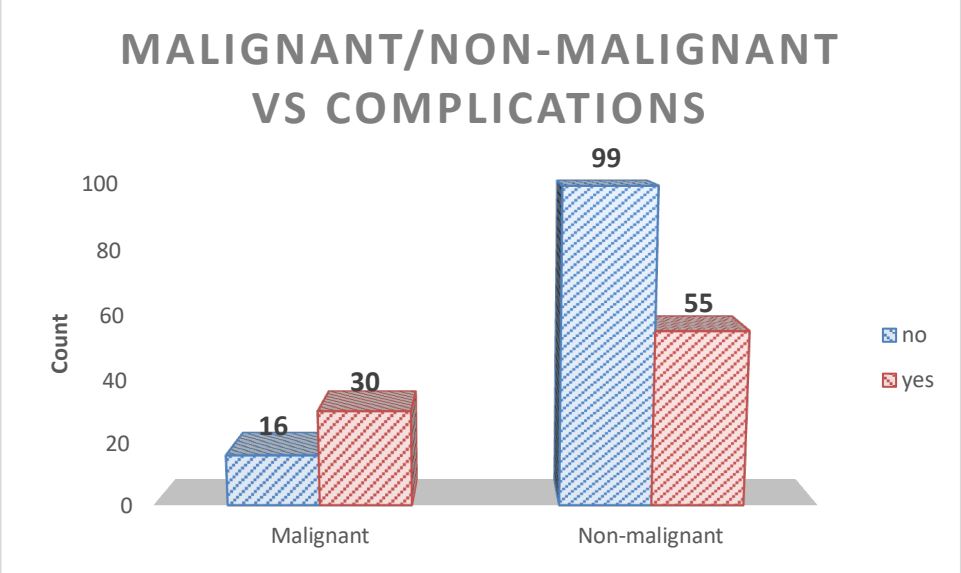
Disease	Count	Percentage
Malignant	46	23.00%
Non-malignant	154	77.00%
Grand Total	**200**	**100.00%**



In this study, 46 patients had malignant disease and 154 patients had non-malignant disease.

Table 11 MALIGNANT/ NON-MALIGNANT VERSUS COMPLICATIONS

Disease	Complications		
	No	Yes	Grand Total
Malignant	16	30	46
Non-malignant	99	55	154
Grand Total	**115**	**85**	**200**

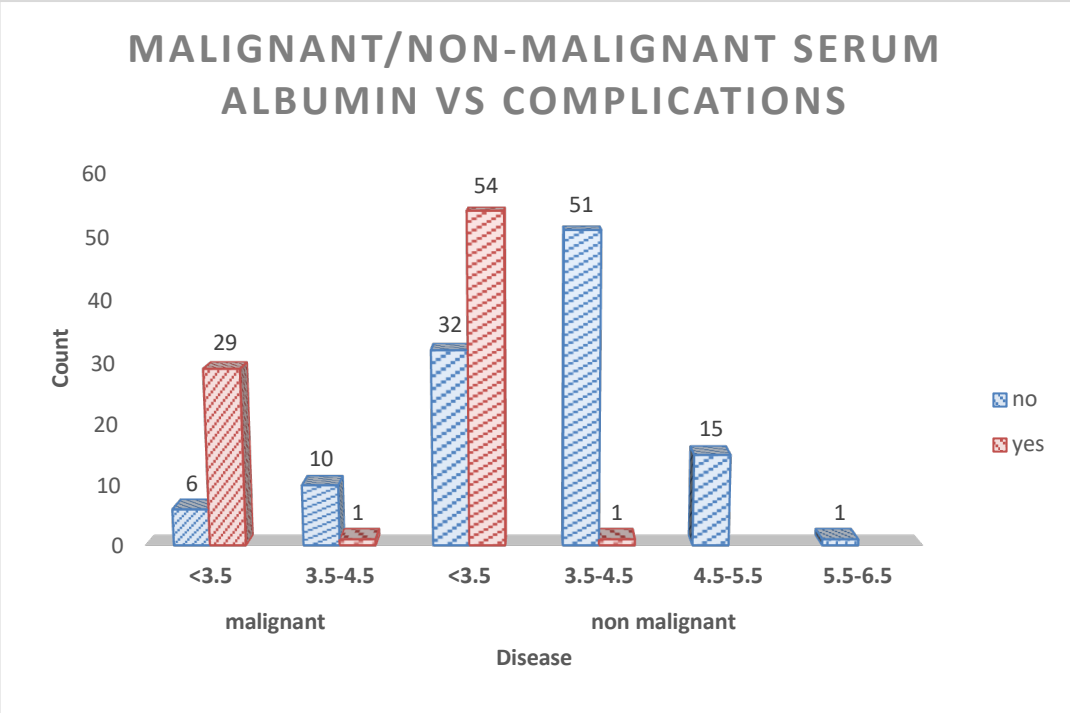


In the malignant patients, 30 patients had post-operative complications and 16 did not have any post-operative complications.

In the non-malignant patients, 99 patients had no post-operative complications and 55 patients had post-operative complications.

Table 12 MALIGNANT/ NON-MALIGNANT SERUM ALBUNIN VERSUS COMPLICATIONS

Disease	Serum Albumin (gm/dL)	Complications		
		No	Yes	Grand Total
Malignant	<3.5	6	29	35
	3.5-4.5	10	1	11
Non-malignant	<3.5	32	54	86
	3.5-4.5	51	1	52
	4.5-5.5	15	0	15
	5.5-6.5	1	0	1
Grand Total		**115**	**85**	**200**



In the malignant patients with complications, 29 patients had serum albumin <3.5 gm/dL and 1 patient had serum albumin 3.5-4.5 gm/dL.

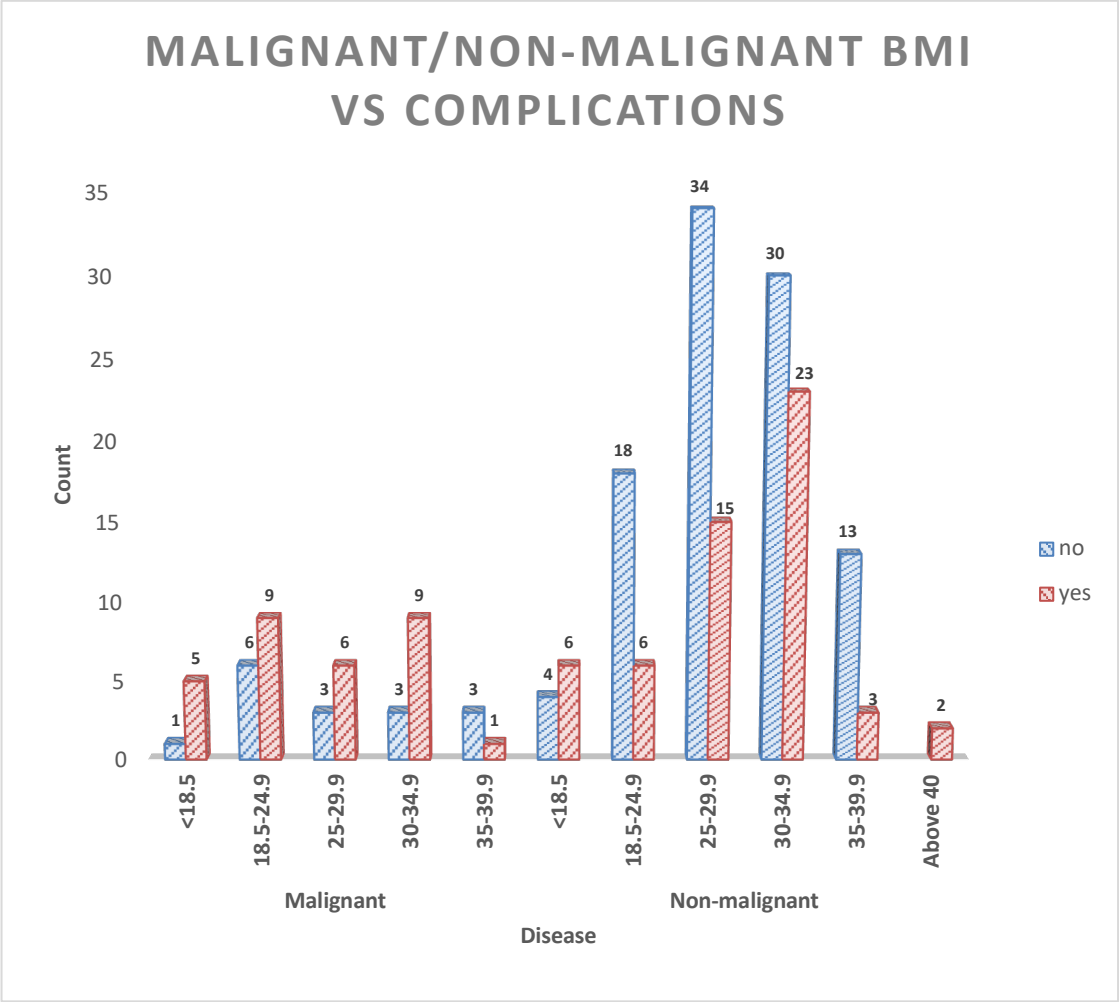
In the malignant patients without complications, 6 patients had serum albumin <3.5 gm/dL and 10 patients had serum albumin 3.5-4.5 gm/dL.

In the non-malignant patients with complications, 54 patients had serum albumin <3.5 gm/dL and 1 patient had serum albumin 3.5-4.5 gm/dL.

In the non-malignant patients without complications, 32 patients had serum albumin <3.5 gm/dL, 51 patients had serum albumin 3.5-4.5 gm/dL, 15 patients had serum albumin 4.5-5.5 gm/dL and 1 patient had serum albumin 5.5-6.5 gm/dL.

Table 13 MALIGNANT/ NON-MALIGNANT BMI Vs COMPLICATIONS

Disease		Complications		
	BMI (Kg/m2)	No	Yes	Grand Total
Malignant	<18.5	1	5	6
	18.5-24.9	6	9	15
	25-29.9	3	6	9
	30-34.9	3	9	12
	35-39.9	3	1	4
Non-malignant	<18.5	4	6	10
	18.5-24.9	18	6	24
	25-29.9	34	15	49
	30-34.9	30	23	53
	35-39.9	13	3	16
	Above 40	0	2	2
Grand Total		**115**	**85**	**200**



In malignant patients with Complications, 5 patients had <18.5 Kg/m^2 BMI, 9 patients had 18.5-24.9 Kg/m^2 BMI, 6 patients had 25-29.9 Kg/m^2 BMI, 9 patients had 30-34.9 Kg/m^2 BMI and 1 patient had 35-39.9 Kg/m^2 BMI.

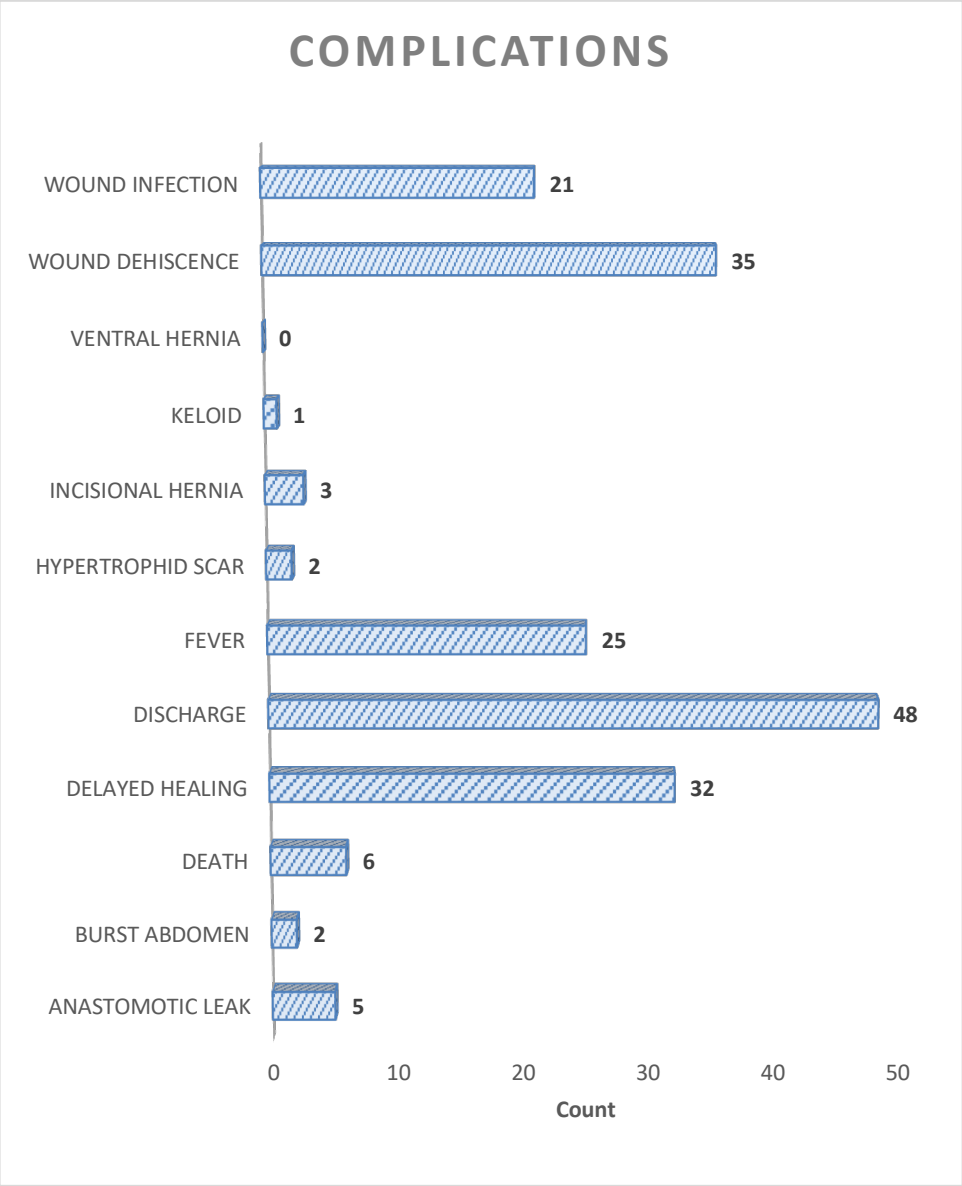
In non-malignant patients with Complications, 6 patients had <18.5 Kg/m^2 BMI, 6 patients had 18.5-24.9 Kg/m^2 BMI, 15 patients had 25-29.9 Kg/m^2 BMI, 23 patients had 30-34.9 Kg/m^2 BMI, 3 patient had 35-39.9 Kg/m^2 BMI and 1 patient had above 40 Kg/m^2 BMI.

In malignant patients without Complications, 1 patient had <18.5 Kg/m^2 BMI, 6 patients had 18.5-24.9 Kg/m^2 BMI, 3 patients had 25-29.9 Kg/m^2 BMI, 3 patients had 30-34.9 Kg/m^2 BMI and 3 patients had 35-39.9 Kg/m^2 BMI.

In non-malignant patients without Complications, 4 patients had <18.5 Kg/m^2 BMI, 18 patients had 18.5-24.9 Kg/m^2 BMI, 34 patients had 25-29.9 Kg/m^2 BMI, 30 patients had 30-34.9 Kg/m^2 BMI and 13 patients had 35-39.9 Kg/m^2 BMI.

Table 14 POST-OPERATIVE COMPLICATIONS

Complications	Count	Percentage
ANASTOMOTIC LEAK	5	2.78%
BURST ABDOMEN	2	1.11%
DEATH	6	3.33%
DELAYED HEALING	32	17.78%
DISCHARGE	48	26.67%
FEVER	25	13.89%
HYPERTROPHID SCAR	2	1.11%
INCISIONAL HERNIA	3	1.67%
KELOID	1	0.56%
VENTRAL HERNIA	0	0.00%
WOUND DEHISCENCE	35	19.44%
WOUND INFECTION	21	11.67%
Grand Total	**180**	**100.00%**



In our study, we found that out of the 200 patients, 115 patients had no post-operative complications and 85 patients had single or a number of post-operative complications.

Among these post-operative complications, 48 patients had wound discharge, 35 patients had wound dehiscence, 32 patients had delayed wound healing, 25 patients had fever, 21 patients had wound infection, 5 patients had anastomotic leak, 3 patients developed incisional hernia, 2 patients had burst abdomen, 2 patients developed hypertrophied scar and 1 patient developed keloid.

6 patients had mortality in the post-operative period.

DISCUSSION AND CONCLUSION

DISCUSSION

Identification of patients who are more likely to experience postoperative problems depends on nutritional assessment. It has been discovered that a number of nutritional markers are useful in predicting patient outcome. For nutritional assessment in this study, preoperative serum albumin level was employed.

Maria W et al studied that patients undergoing gastrointestinal surgery are following perioperative treatment in enhanced recovery after surgery (ERAS) protocols. Although oral feeding is supposed not to be stopped perioperatively with respect to ERAS, malnourished patients and inadequate calorie intake are common. Malnutrition, even in overweight or obese patients, is often underestimated. Patients at metabolic risk have to be identified early to confirm the indication for nutritional therapy. The monitoring of nutritional status postoperatively has to be considered in the hospital and after discharge, especially after surgery in the upper gastrointestinal tract, as normal oral food intake is decreased for several months.[67]

Jeong MS et al patients undergoing major gastrointestinal (GI) surgery, particularly those with malignancies, have a high risk for malnutrition, requiring perioperative nutritional support to reduce complications. 536 patients from 83 hospitals, mean age 58.8 ± 15.1 years, 59.1% males, were eligible. Leading diagnosis were GI diseases (48.7%) and GI cancer (45.9%). Malnutrition risk was moderate to high in 54% of patients, low in 46%. Hospital length of stay (LOS) before the NID was 9.3 ± 19.0 days, and time since last surgery 3.7 ± 2.4 days. Lowest caloric/protein deficits were observed in patients receiving EN + PN, followed by PN alone and EN alone. Type of clinical nutrition, Body Mass Index and LOS on surgical intensive care unit (SICU) and/or surgical ward were independent predictors of caloric and of protein deficit. There was a high prevalence of postoperative nutritional deficits in Asian GI surgery patients, who are either preoperatively malnourished or at risk of malnutrition, indicating a need to improve nutritional support and education.[68]

Byakhodi KG et al showed that wound healing requires energy and is a catabolic process. Patients who are severely malnourished demonstrate impaired wound healing and predisposition to infection. They also suffer deficient immune mechanisms. The degree of malnutrition is estimated on the basis of weight loss during the past 6 months, physical findings and plasma protein assessment. The purpose of this study was to investigate serum albumin

and BMI as predictors of postoperative mortality and morbidity. Total of 117 patients were included in the study admitted in KIMS Hubli from November 2012 to June 2014.

The present study was a Prospective Observational study. This Study was conducted from July 2021 to June 2022 at Department of General Surgery, Santosh Medical College & Hospital Ghaziabad. Total 200 patients were included in this study. Among these patients, 115 patients (57.5%) did not have any post-operative complications whereas 85 patients (42.5%) had one or a group of complications.

1) Age –

In our study, most of the patients (58 patients, 29%) were in 40-49 years of age. Minimum age was 18 years and maximum age was 80 years. The mean age was 43.3 years with standard deviation of 14.73.

We found that, most patients with complications fell in the age group of 40-49 years (23 patients, 39.65%). Age was statistically significant with Complications (p=0.0154).

2) Gender –

We found that, male population (121 patients, 60.5%) was higher than the female population (79 patients, 39.5%).

In these, 52 male patients (42.97%) had post-operative complications and 33 female patients (41.77%) had post-operative complications but this was not statistically significant (p=0.5996).

3) BMI –

In our study, most patients had BMI of 30-34.9 Kg/m^2 (65 patients, 32.5%). Lowest BMI was 15.9 Kg/m2 and highest BMI was 46.6 Kg/m^2. Mean BMI was 27.41 Kg/m^2 and standard deviation was 5.645.

Among these, patients with BMI <18.5 Kg/m^2 had the highest post-operative complication rate (68.75%) followed by BMI 30-34.9 Kg/m^2 (49.23%). The mean difference of BMI (Kg/m^2) - Complications was 26.9825 with 95% confidence interval [26.1892 to 27.7758]. The difference of mean BMI (Kg/m^2) - Complications was statistically significant (p<.001).

Mullen et al reported that in the post operative period being underweight was associated with increased mortality and obese individuals had more wound complication.

Azodi concluded that a BMI of 27.5 kg/m^2 or more was associated with more post-operative complication after open appendicectomy in patients with non-perforated appendicitis ($p<0.001$).[69]

4) <u>Serum Albumin</u> –

We found that most of the patients had serum albumin <3.5 gm/dL (121 patients, 60.5%). Minimum serum albumin observed was 2.0 gm/dL and maximum serum albumin observed was 6.20 gm/dL. Mean serum albumin was 3.415 gm/dL and standard deviation was 0.7333.

Out of these, patients with serum albumin <3.5 gm/dL had most complications (68.59%) as compared to those with serum albumin >3.5 gm/dL (2.5%). The mean difference of Serum Albumin (gm/dL) - Complications was 2.98955 with 95% confidence interval [2.83006 to 3.14904]. The difference of mean BMI (Kg/m2) - Complications was statistically significant (p<.001).

Gibbs et al observed that a decrease in Serum Albumin from concentration greater than 4.6 g/dL to less than 2.1 mg/dL ($p<0.001$) was associated with exponential increase in morbidity and mortality and that it was a good prognostic indicator, whereas anthropometric markers could not predict postoperative outcome.[70]

According to Foley et al post operative complication rate was higher when albumin was lower than 2.5 g/dl ($p<0.001$).[71]

According to Beghetto et al it was concluded that serum albumin level was the strongest predictive parameter for death and hospital infection (<3.5g/dL).

Engelman et al observed that albumin less than 2.5 gm/dL ($p<0.001$) and BMI less than 20 kg/m^2 ($p<0.005$) and greater than 30 kg/m^2 ($p<0.005$) was associated with increase in post-operative complications.

Lalhruaizela S et al (2020) observed that the purpose of nutritional support in the surgical patient is to prevent or reverse the catabolic effects of disease or injury. Most of the

early post-operative complications were present in patients with serum albumin level 2.1-2.7 gm/dL (43.43%). The relation between pre-operative serum albumin and early and late post-operative complications was found to be statistically significant. There was a significant co-relation between age, BMI and complications. There were four deaths in the study. Pre-operative Hypoalbuminemia <3.0 gm/dL is a strong and independent risk factor for post-operative morbidity and mortality in GI surgeries and BMI is a strong indicator for post-operative complications.[4]

Mozumder MN et al Mean BMI was lower in group B (20.88 ± 3.71 kg/m2) than in group A (22.26 ± 1.67 kg/m2), which was found statistically significant (p=0.006). In terms of postoperative outcome, total chest drain collection was significantly higher in group B (968.80 ± 183.49 ml) than in group A (816.00 ± 113.40 ml), (p=0.001). Duration of ICU stay were significantly longer in group B (4.60 ± 0.76 days) than in group A (3.92 ± 0.86 days) (p=0.005). Pearson co-efficient correlation test showed strong inverse relationship of serum albumin with total chest drain, ICU stay and hospital stay following valve replacement surgery (r=-0.473, r=-0.448 & r=-0.487 respectively), which was most significant than age and BMI ($p \leq 0.001$). Multivariate logistic regression analysis was done to assess the predictive value of serum albumin level, age and BMI, where preoperative serum albumin level was found to be the most valuable predictor of postoperative outcome after valve replacement surgery (B=-2.251, OR 0.105, 95% CI 0.011-0.986, p<0.05).[64]

Study Name	Serum Albumin (gm/dL) associated with increased complications	P value
Beghetto et al	<3.5	<0.05
Leite et al	<3	<0.05
Brown et al	<3	<0.05
Engelman et al	<2.5	<0.001
Foley et al	<2.5	<0.001
Present Study	**<3.5**	**<0.001**

Table 15 SUMMARY OF VARIOUS STUDIES

5) <u>Malignant Disease</u> –

We observed that, 46 patients (26 males and 20 females) in our study had a malignant disease (23%). Among these, 30 patients had post-operative complications (65.21%).

Most patients with malignant disease had BMI of 18.5-24.9 Kg/M^2 (15 patients, 32.60%). Lowest BMI was 17.3 Kg/m2 and highest BMI was 36 Kg/m^2. Complication rate was higher in patients with BMI <18.5 Kg/m2(83.33%). Complication rate among the patients with non-malignant disease was lower (35.71%). The mean difference of BMI (Kg/m^2) - Complications was 25.0348 with 95% confidence interval [23.1650 to 26.9046]. The difference of mean BMI (Kg/m^2) - Complications was statistically significant (p<.001).

Also, most patients with malignant disease had serum albumin of <3.5 gm/dL (29 patients, 63.04%). Minimum serum albumin observed was 2.0 gm/dL and maximum serum albumin observed was 4.21 gm/dL. Complication rate was higher in patients with serum albumin <3.5 gm/dL (82.85%). Complication rate among the patients with non-malignant disease was lower (35.71%). The mean difference of Serum Albumin (gm/dL) - Complications was 2.42413 with 95% confidence interval [2.15252 to 2.69574]. The difference of mean BMI (Kg/m2) - Complications was statistically significant (p<.001).

Sean LG et al (2015) The largest study compared the in-hospital mortality in 7227 patients who underwent oesophageal surgery for malignancy. The percentage of in-hospital mortality was associated with low serum albumin (<15.0 vs >35.0 g/l, 21.0 vs 11.3%, P <0.001).[72]

Babu CS et al (2022) Out of 50 patients studied 18 (36%) were malignant and 32 (64%) were non-malignant. 20 patients developed complications, 4 (20%) were malignant and 16 (80%) were non-malignant. Among the patients with malignancy (18) only 4 had complications, of which 2 (11%) were <3.0 g/dl and 2 (11%) between 3.1- 3.5 g/dl. Albumin less than 2.5 g/dl (p<0.001) and BMI less than 20 Kg/m2 (p<0.005) and greater than 30 Kg/m2 (p <0.005) was associated with increase in post-operative complications. The correlation between the serum albumin and complication rate was statistically significant in the malignant diseases when considered separately.[73]

CONCLUSION

1. The majority of the patients in our study were between the ages of 40 and 49 years. Age and complications had statistically significant associations.

2. The male to female ratio was 1.53:1, although this was not statistically significant because the male population was larger than the female population.

3. According to our study, problems were common among malnourished patients with BMIs below 18.5 kg/m^2 and obese-I category patients with BMIs between 30 and 34.9 kg/m^2. With complications, BMI was statistically significant.

4. It was discovered that the majority of individuals with complications had serum albumin levels that were lower than 3.5 gm/dL. Statistically, it was significant.

5. When compared to individuals with non-malignant disease, we found that patients with malignant disease typically had low BMIs (18.5 Kg/m^2), low serum albumin levels (3.5 g/dL), and a greater complication rate which was statistically significant.

We came to the conclusion that there was a substantial relationship between pre-operative serum albumin and post-operative morbidity and mortality in major general surgeries, as well as between the nutritional state of the patient as evaluated by plasma albumin level.

BIBLIOGRAPHY

1. Ramole Y, Chaudhary A, Patel BP, Divan AS, Jain A, Songra MC. Assessment of preoperative serum albumin level and its correlation with postoperative wound complication in major elective abdominal surgeries. International Surgery Journal [Internet] 2020 [cited 2022 Oct 16];7(5):1508–11. Available from: https://www.ijsurgery.com/index.php/isj/article/view/5671
2. Bhagvat V, Ghetla S, Shetty T, Upwanshi M. Role of serum albumin and body mass index as predictors of post-operative morbidity and mortality in elective major abdominal surgeries. International Surgery Journal 2017;
3. Lohe Y, V. S, Sharma R, Lombi N, M. H, G. A, et al. Role of serum albumin and body mass index in the outcome of major elective abdominal surgery. International Surgery Journal [Internet] 2019 [cited 2022 Oct 16];6(4):1165–71. Available from: https://www.ijsurgery.com/index.php/isj/article/view/4122
4. Lalhruaizela S, Lalrinpuia B, Gupta VD. Serum Albumin is a Predictor for Postoperative Morbidity and Mortality in Gastrointestinal Surgeries. JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH 2020;
5. Protein–energy malnutrition occurs as a result of a relative or absolute deficiency of energy and protein. It may be primary, due to inadequate food intake, - ppt download [Internet]. [cited 2022 Oct 16];Available from: https://slideplayer.com/slide/9386627
6. Moman RN, Gupta N, Varacallo M. Physiology, Albumin. StatPearls 2022;32(6).
7. Moman RN, Varacallo M. Physiology, Albumin. 2019.
8. Astapenko D, Pouska J, Škola J, Beneš J, Náhlovský J, Mynář M, et al. Use of albumin in perioperative and intensive care medicine. Anesteziologie a Intenzivni Medicina 2020;31(3).
9. Doweiko JP, Nompleggi DJ. The role of albumin in human physiology and pathophysiology, part III: Albumin and disease states. Journal of Parenteral and Enteral Nutrition1991;15(4).
10. Caironi P, Langer T, Gattinoni L. Albumin in critically ill patients: The ideal colloid? Curr Opin Crit Care2015;21(4).
11. Belinskaia DA, Voronina PA, Shmurak VI, Vovk MA, Batalova AA, Jenkins RO, et al. The universal soldier: Enzymatic and non-enzymatic antioxidant functions of serum albumin. Antioxidants2020;9(10).
12. Soeters PB, Wolfe RR, Shenkin A. Hypoalbuminemia: Pathogenesis and Clinical Significance. Journal of Parenteral and Enteral Nutrition2019;43(2).
13. Wiedermann CJ. Hypoalbuminemia as surrogate and culprit of infections. Int J Mol Sci2021;22(9).
14. The Relationship Between Serum Albumin Level and Nutritional Parameters with Mortality Rate, For Icu-Covid-19 Patients at Hazem Mebaireek General Hospital (HMGH), State of Qatar (March to September / 2020). Food Science and Quality Management 2021;

15. Eckart A, Struja T, Kutz A, Baumgartner A, Baumgartner T, Zurfluh S, et al. Relationship of Nutritional Status, Inflammation, and Serum Albumin Levels During Acute Illness: A Prospective Study. American Journal of Medicine 2020;133(6).
16. Galata C, Busse L, Birgin E, Weiß C, Hardt J, Reißfelder C, et al. Role of Albumin as a Nutritional and Prognostic Marker in Elective Intestinal Surgery. Can J Gastroenterol Hepatol 2020;2020.
17. Strauss RS, Dietz WH. Community and International Nutrition Low Maternal Weight Gain in the Second or Third Trimester Increases the Risk for Intrauterine Growth Retardation 1. J Nutr 1999;129.
18. Garcovich M, Zocco MA, Gasbarrini A. Clinical use of albumin in hepatology. Blood Transfusion2009;7(4).
19. Caraceni P, Tufoni M, Bonavita ME. Clinical use of albumin. Blood Transfusion2013;11(SUPPL. 4).
20. Martelli A, Strada P, Cagliani I, Brambilla G. Guidelines for the Clinical Use of Albumin: Comparison of Use in Two Italian Hospitals and a Third Hospital Without Guidelines. Curr Ther Res Clin Exp 2003;64(9).
21. Puzio TJ, Kozar RA. Nutrition in the critically ill surgical patient. Curr Opin Crit Care2020;26(6).
22. Yeh DD, Martin M, Sakran J v., Meier K, Mendoza A, Grant AA, et al. Advances in nutrition for the surgical patient. Curr Probl Surg 2019;56(8).
23. Khan M, Latifi R. Nutrition in surgical patients: How soon is too soon? Curr Opin Crit Care2019;25(6).
24. Gillis C, Wischmeyer PE. Pre-operative nutrition and the elective surgical patient: why, how and what? Anaesthesia2019;74.
25. Ortiz LA, Dante Yeh D. Nutrition in the post-surgical patient: Myths and misconceptions. Curr Surg Rep 2017;5(7).
26. Roshni PR, Purushothaman A. Role of total parenteral nutrition in surgical patients. Int J Pharm Sci Rev Res 2015;33(2).
27. Eirmann L. Nutritional Assessment. Veterinary Clinics of North America - Small Animal Practice2016;46(5).
28. Reber E, Gomes F, Vasiloglou MF, Schuetz P, Stanga Z. Nutritional risk screening and assessment. J Clin Med2019;8(7).
29. Pimenta FS, Oliveira CM, Hattori WT, Teixeira KR. Agreement between Subjective Global Nutritional Assessment and the nutritional assessment of the World Health Organization. J Pediatr (Rio J) 2018;94(6).
30. Spriet LL. New insights into the interaction of carbohydrate and fat metabolism during exercise. Sports Medicine 2014;44(SUPPL.1).
31. Leong SY, Duque SM, Muhammad Abduh SB, Oey I. Carbohydrates. In: Innovative Thermal and Non-Thermal Processing, Bioaccessibility and Bioavailability of Nutrients and Bioactive Compounds. 2019.
32. Wilkinson DJ. Historical and contemporary stable isotope tracer approaches to studying mammalian protein metabolism. Mass Spectrom Rev2018;37(1).

33. Viana M v., Becce F, Pantet O, Schmidt S, Bagnoud G, Thaden JJ, et al. Impact of β−hydroxy-β−methylbutyrate (HMB) on muscle loss and protein metabolism in critically ill patients: A RCT. Clinical Nutrition 2021;40(8).
34. Remmerie A, Scott CL. Macrophages and lipid metabolism. Cell Immunol 2018;330.
35. Chung KW. Advances in understanding of the role of lipid metabolism in aging. Cells2021;10(4).
36. Schoeler M, Caesar R. Dietary lipids, gut microbiota and lipid metabolism. Rev Endocr Metab Disord2019;20(4).
37. Hirotsu C, Tufik S, Andersen ML. Interactions between sleep, stress, and metabolism: From physiological to pathological conditions. Sleep Science2015;8(3).
38. Wang PH, Huang BS, Horng HC, Yeh CC, Chen YJ. Wound healing. Journal of the Chinese Medical Association2018;81(2).
39. Mathew-Steiner SS, Roy S, Sen CK. Collagen in wound healing. Bioengineering2021;8(5).
40. Qing C. The molecular biology in wound healing & non-healing wound. Chinese Journal of Traumatology - English Edition2017;20(4).
41. Krzyszczyk P, Schloss R, Palmer A, Berthiaume F. The role of macrophages in acute and chronic wound healing and interventions to promote pro-wound healing phenotypes. Front Physiol2018;9(MAY).
42. Cho YD, Kim KH, Lee YM, Ku Y, Seol YJ. Periodontal wound healing and tissue regeneration: A narrative review. Pharmaceuticals2021;14(5).
43. McDonald S, Bott A. Surgical site infections. Surgery (United Kingdom)2020;38(3).
44. Reeves N, Torkington J. Prevention of surgical site infections. Surgery (United Kingdom)2022;40(1).
45. Jaiswal NK, Shekhar S. Study of burst abdomen: it's causes and management. International Surgery Journal 2018;5(3).
46. Ling ML, Apisarnthanarak A, Abbas A, Morikane K, Lee KY, Warrier A, et al. APSIC guidelines for the prevention of surgical site infections. Antimicrob Resist Infect Control 2019;8(1).
47. Yang X, Aihemaiti M, Zhang H, Jiang L, Zhang G, Qin M, et al. Mesh-preservation approach to treatment of mesh infection after large incisional ventral hernia repair—how I do it. Ann Transl Med [Internet] 2019 [cited 2022 Nov 7];7(22):698–698. Available from: https://atm.amegroups.com/article/view/31249/html
48. National Healthcare Safety Network. Surgical Site Infection Event (SSI). Centers for Disease Control and Prevention 2021;(January).
49. Birhanu Y, Endalamaw A. Surgical site infection and pathogens in Ethiopia: A systematic review and meta-analysis. Patient Saf Surg2020;14(1).
50. Adogwa O, Martin JR, Huang K, Verla T, Fatemi P, Thompson P, et al. Preoperative serum albumin level as a predictor of postoperative complication after spine fusion. Spine (Phila Pa 1976) 2014;39(18).
51. Bhandari TR, Shahi S, Bhandari RS, Lakhey PJ. Preoperative serum albumin level as a predictor of perioperative outcome in patient undergoing major gastrointestinal surgery. Journal of Society of Surgeons of Nepal 2016;19(2).

52. Labgaa I, Joliat GR, Kefleyesus A, Mantziari S, Schäfer M, Demartines N, et al. Is postoperative decrease of serum albumin an early predictor of complications after major abdominal surgery? A prospective cohort study in a European centre. BMJ Open 2017;7(4).
53. Chiang JM, Chang CJ, Jiang SF, Yeh CY, You JF, Hsieh PS, et al. Pre-operative serum albumin level substantially predicts post-operative morbidity and mortality among patients with colorectal cancer who undergo elective colectomy. Eur J Cancer Care (Engl) 2017;26(2).
54. Inagaki E, Farber A, Eslami MH, Kalish J, Rybin D v., Doros G, et al. Preoperative hypoalbuminemia is associated with poor clinical outcomes after open and endovascular abdominal aortic aneurysm repair. In: Journal of Vascular Surgery. 2017.
55. Sharma L, Purohit PM, Pipal D, Kothari S, Srivastava H, Soni A. Serum albumin as predictor of post-operative morbidity and mortality. International Surgery Journal 2017;4(3).
56. Y. W, X. L, Y. J, H. T, X. L, N. L, et al. Preoperative serum albumin level as a predictor of postoperative pneumonia after femoral neck fracture surgery in a geriatric population. Clin Interv Aging 2019;14.
57. Wan Y, Wang Z, Wang J, Su H, Guo X, Wu J, et al. Genetic parameters of the thick-to-thin albumen ratio and egg compositional traits in layer-type chickens. Br Poult Sci 2019;60(5).
58. Byakhodi DrKG, Kamat DrV v, Sunil DrC. A prospective study to predict post-operative morbidity and mortality in emergency abdominal surgeries using preoperative serum albumin and BMI. International Journal of Surgery Science 2019;3(3).
59. Issangya CE, Msuya D, Chilonga K, Herman A, Shao E, Shirima F, et al. Perioperative serum albumin as a predictor of adverse outcomes in abdominal surgery: Prospective cohort hospital based study in Northern Tanzania. BMC Surg 2020;20(1).
60. P DrA, Vikraman DrB, C P DrH, Chakiath DrJA, Perumbilavil DrG, C Tenny DrT. Clinical Correlation between Preoperative Serum Albumin and Postoperative Outcome in Major Gastrointestinal Surgeries. Saudi Journal of Medicine 2020;05(03).
61. Rathod SD, Wagh DD. A Prospective Study to Correlate Preoperative Serum Albumin as a Predictor of Post-Operative Mortality and Morbidity. Original Research Article J Evolution Med Dent Sci 2021;10.
62. Reis TG, Silva RAWP da, Nascimento E dos S, Bessa J de, Oliveira MC, Fava AS, et al. Early postoperative serum albumin levels as predictors of surgical outcomes in head and neck squamous cell carcinoma. Braz J Otorhinolaryngol [Internet] 2021 [cited 2022 Oct 16];Available from: https://pubmed.ncbi.nlm.nih.gov/33875388/
63. Liu Q, Gao K, Zheng C, Guo C. The Risk Factors for Perioperative Serum Albumin Variation in Pediatric Patients Undergoing Major Gastroenterology Surgery. Front Surg [Internet] 2020 [cited 2022 Oct 16];7:627174. Available from: /pmc/articles/PMC7873733/
64. Mozumder MdNEE, Rahman MdM, Hoque MdR, Alauddin Md, Khan OS, Morshed AJMdT, et al. Preoperative Serum Albumin Level as a Predictor of Outcome after Valve Replacement Surgery. Journal of Cardiac and Pulmonary Rehabilitation [Internet] 2021

[cited 2022 Oct 16];5(2):1–5. Available from: https://www.omicsonline.org/open-access/preoperative-serum-albumin-level-as-a-predictor-of-outcome-after-valve-replacement-surgery-116041.html

65. Larsen PB, Liest S, Hannani D, Jørgensen HL, Sørensen LT, Jørgensen LN. Preoperative Hypoalbuminemia Predicts Early Mortality Following Open Abdominal Surgery in Patients Above 60 Years of Age. Scand J Surg [Internet] 2021 [cited 2022 Oct 16];110(1):29–36. Available from: https://pubmed.ncbi.nlm.nih.gov/31769347/
66. Silahli M, Tekin M. Albumin Haemoglobin Index: A Novel Pre-operative Marker for Predicting Mortality and Hospital Stay in Patients Under One-Year Undergoing Gastrointestinal Surgeries. Afr J Paediatr Surg [Internet] 2022 [cited 2022 Oct 16];19(2):89. Available from: /pmc/articles/PMC8809471/
67. Wobith M, Weimann A. Oral nutritional supplements and enteral nutrition in patients with gastrointestinal surgery. Nutrients2021;13(8).
68. Seo JM, Joshi R, Chaudhary A, Hsu HS, Trung LV, Inciong JF, et al. A multinational observational study of clinical nutrition practice in patients undergoing major gastrointestinal surgery: The Nutrition Insights Day. Clin Nutr ESPEN 2021;41.
69. Azodi OS, Lindström D, Adami J, Bellocco R, Linder S, Wladis A. Impact of body mass index and tobacco smoking on outcome after open appendicectomy. In: British Journal of Surgery. 2008.
70. Gibbs J. Preoperative Serum Albumin Level as a Predictor of Operative Mortality and Morbidity. Archives of Surgery 1999;134(1).
71. Foley EF, Borlase BC, Dzik WH, Bistrian BR, Benotti PN. Albumin Supplementation in the Critically Ill: A Prospective, Randomized Trial. Archives of Surgery 1990;125(6).
72. Goh SL, de Silva RP, Dhital K, Gett RM. Is low serum albumin associated with postoperative complications in patients undergoing oesophagectomy for oesophageal malignancies? Interact Cardiovasc Thorac Surg 2015;20(1).
73. Welsford MR, Johnson SD, Pérez F, Schrey NM, SCHREY AW, Heist EJEJ, et al. A convergência espacial do conhecimento em Portugal. Mol Ecol 2008;17(1).

FIGURES

Figure 1 A MOLECULE OF ALBUMIN..... 7

Figure 2 SYNTHESIS OF ALBMIN 8

Figure 3 ANASTOMOTIC LEAK..... 18

Figure 4 PATHWAYS FOR THE CATABOLISM 25

Figure 5 GLUCOSE METABOLISM 26

Figure 6 PROTEIN METABOLISM..... 28

Figure 7 FATE OF DIETARY PROTEIN..... 29

Figure 8 LIPID METABOLISM..... 30

Figure 9 FUNCTIONS OF PLASMA PROTEIN..... 31

Figure 10 TIME COURSE OF CHANGES IN ACUTE PHASE PROTEINS..... 33

Figure 11 METABOLIC RESPONSE TO TRAUMA 37

Figure 12 STAGES OF WOUND HEALING..... 39

Figure 13 PHASES OF WOUND HEALING 43

Figure 14 DIAGRAMATIC PRESENTATION STAGES OF WOUND HEALING 44

Figure 15 CLASSIFICATION OF WOUND HEALING 46

Figure 16 ROLE OF NUTRIENTS IN WOUND HEALING 49

Figure 17 CLASSIFICATION OF SURGICAL SITE INFECTION..... 50

Figure 18 SUPERFICIAL INCISIONAL SSI 51

Figure 19 DEEP INCISIONAL SSI..... 52

Figure 20 PURULENT DISCHARGE FROM BURST ABDOMEN……...53

Figure 21 INFECTED HERNIA MESH…….54

TABLES

Table 1 AGE DISTRIBUTION 76

Table 2 GENDER DISTRIBUTION 77

Table 3 DISTRIBUTION OF BMI 78

Table 4 DISTRIBUTION OF SERUM ALBUMIN 79

Table 5 POST-OPERATIVE OUTCOME 80

Table 6 AGE VERSUS COMPLICATIONS 81

Table 7 GENDER VERSUS COMPLICATIONS 83

Table 8 BMI VERSUS COMPLICATIONS 84

Table 9 SERUM ALBUMIN VERSUS COMPLICATIONS 86

Table 10 MALIGNANT VERSUS NON-MALIGNANT 87

Table 11 MALIGNANT/ NON-MALIGNANT VERSUS COMPLICATIONS 88

Table 12 MALIGNANT/ NON-MALIGNANT S. ALBUNIN Vs COMPLICATIONS 89

Table 13 MALIGNANT/ NON-MALIGNANT BMI Vs COMPLICATIONS 91

Table 14 POST-OPERATIVE COMPLICATIONS 93

Table 15 SUMMARY OF VARIOUS STUDIES 99

PROFORMA

Serial No.: Date:

Name: Age/Sex:

IPD no.: Occupation:

Address:

Final Diagnosis:

Treatment Plan: Date of surgery:

HISTORY

Chief complaints:

History of presenting illness:

Past history:

Family history:

Co morbidities:

EXAMINATION

General physical examination

Height(m): Weight (Kg): BMI(Kg/m^2):

Pallor: Icterus: Cyanosis: Clubbing: Lymphadenopathy: Edema:

Systemic examination

- CVS:
- RS:
- P/A:
- CNS:

Local Examination:

INVESTIGATIONS:

Haemoglobin(gm%): Blood sugar(mg/dl): RBS:

Viral Markers: FBS: PPBS:

LFT:

S. Albumin(gm/dl):

Serum Urea(mg/dl): Serum Creatinine(mg/dl):

Urine (R/M):

OPERATION DETAILS:

Duration:

Findings:

DAY OF DISCHARGE:

FOLLOW UP:

- Fever
- Wound infection and discharge
- Wound dehiscence
- Anastomotic leak
- Burst abdomen
- Delayed wound healing
- Complications of scar – Keloid/ Hypertrophic Scar
- Ventral hernia
- Death

SANTOSH MEDICAL COLLEGE AND HOSPITAL (GHAZIABAD)

INFORMED CONSENT

I _____ Son/Daughter/Wife of _____ resident of _____hereby declare that I give my full, free and voluntary informed consent in the thesis - "**PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES".** Dr._____ has informed me to my full satisfaction, in the language I understand, about the purpose, nature of study and various laboratory investigations for the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.

I give full consent for being enrolled in the above study and I reserve my rights to withdraw from this study, whenever I wish without prejudice of my right to undergo further treatment at Santosh Hospital, Ghaziabad U.P.

Patient's/ Patient's relative's signature
Or thumb impression

Doctors signature

Name:

Date:

We have witnessed that the patient signed the above form in the presence of his free will after fully understanding its content.

Signature of Witness

Name

Signature of Investigator
Name: Dr. Abhinav Mone
Designation: PG Department of General Surgery
Contact No.- 09833992911

संतोष मेडिकल कॉलेज एवं अस्पताल (गाजियाबाद)

<u>सहमति पत्र (सूचित)</u>

मैं _____ बेटा/ बेटी/ पत्नी_____निवासी _____ घोषणा करता हूं कि डॉ ._____, ने मुझे मेरी भाषा में अपने अध्ययन "**PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES"** के उद्देश्य के बारे में, और अध्ययन के दौरान अपनाई जाने वाली विभिन्न प्रयोगशाला जांच के बारे में सूचित कर दिया है। मुझे अध्ययनकी अवधि और उसके दौरान अपनाए जाने वाले निर्देश भी मेरी भाषा मे समझा दिए गए हैं| मैं उपरोक्त अध्ययन में बिना किसी दबाव के दाखिला ले रहा हूं और पूर्ण सहमति दे रहा हूं | सन्तोष अस्पताल, गाजियाबाद में अपने आगे के उपचार पर बिना प्रतिकूल प्रभाव डाले इस अध्ययन से और प्रयोगशाला जांच की प्रकृति से मैं जब चाहूँ बाहर जाने के अपने अधिकार को सुरक्षित रख रहा हूं |

रोगी / रोगी के रिश्तेदार के हस्ताक्षर डॉक्टर के हस्ताक्षर

नाम

दिनांक:

मेरी उपस्थिति में मरीज ने पूरी तरह से डाकटर के बताए गए अध्ययन और निर्देशों को समझने के बादअपनी स्वतंत्र इच्छा से ऊपर के फार्म पर हस्ताक्षर किए हैं ।

हस्ताक्षर

नाम: डॉ अभिनव मोने

पदनाम: पीजी

शल्य चिकित्सा विभाग

संपर्क नंबर- 9340935303

SANTOSH MEDICAL COLLEGE & HOSPITAL

Details of Patient Information Sheet

Human Subject Research Proposal

1) Identifying Data

Name of Researcher: Dr. Abhinav Mone

Department: Department of General Surgery

Title of Project**: PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES.**

2) Description of Human Subjects Safeguards and Rights:

A) Possible psychological or physical risk or discomfort, involved in research

Yes □ NO □ (if no, skip item B)

If Yes, answer the following

Why necessary?

1. To have the common disease
2. Specificity and sensitivity of the treatment

B) Make sure the following questions are answered in your research proposal on collection of Data:

1) When will it take place?

Jan 2021 to June 2022.

2) What length of time needed for complete examination of the patient?

Approx 1 hour.

3) What period of time needed?

Approx 1 year.

4) Which specific place within institution?

Department of Surgery.

5) How much population required including number of subjects?

6) Name of instrument to be used for data collection?

A pre-operative Serum Albumin level as the predictor of post-operative morbidity and mortality in major general surgeries.

7) Methodology to be used (write details)?

- A detailed history of all patients will be taken and a thorough clinical examination will be done. All patients will be analyzed in various aspects like age, sex, body weight, BMI, risk factors, mode of presentation, and laboratory-based evidence. Patients will also be evaluated for other risk factors like obesity, HTN, DM and malignant disease.
- Pre-operative routine investigations like Blood including Serum Albumin, Urine, CxR, and USG will be done and their correlation with the post-operative morbidities or mortality will be established.
- The analyzed data will be compared with other series in literature and discussed. A master chart dealing with all aspects will be designed and presented. A proforma will be used for the study purpose.

C) Will Subjects be given research results if they so request?

Yes □ No □

ETHICAL CLEARANCE

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[48] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES",** submitted by **Dr. Abhinav Mone,** MD/MS student of Batch 2020-2021, Department of **General Surgery**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"PRE-OPERATIVE SERUM ALBUMIN AS A PREDICTOR OF POST-OPERATIVE MORBIDITY AND MORTALITY IN MAJOR GENERAL SURGERIES", Dr. Abhinav Mone** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH DEEMED TO BE UNIVERSITY REGISTRAR GHAZIABAD, NCR DELHI

Dr. Abhinav Mone
MD/MS student of batch 2020-2021
Department of General Surgery
Email: abhinavmone@gmail.com

Copy to:

1. The Vice Chancellor
2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. T.S. Bhagat (Guide)
7. HOD of **General Surgery**

PLAGARISM CERTIFICATE

Ouriginal by Turnitin

Document Information

Analyzed document	Thesis Dr Abhinav Mone (1).docx (D149321961)
Submitted	2022-11-11 06:43:00
Submitted by	Dr Jyoti Batra
Submitter email	drjyotibatra89@gmail.com
Similarity	0%
Analysis address	drjyotibatra89.sug@analysis.urkund.com

Sources included in the report

W URL: https://www.ijsurgery.com/index.php/isj/article/viewFile/4122/2741
Fetched: 2022-11-09 07:55:49 4

Submitted text As student entered the text in the submitted document.
Matching text As the text appears in the source.

1/4 **SUBMITTED TEXT** 21 WORDS **73%** **MATCHING TEXT** 21 WORDS

patients had serum albumin >3.5 gm/dL and 1 patient had serum albumin 3.5-4.5 gm/dL. In the

patients had serum albumin less than 3.5 gm/dl and 122 (81.3%) patients had serum albumin ≥3.5 gm/dl. The

W https://www.ijsurgery.com/index.php/isj/article/viewFile/4122/2741

2/4 **SUBMITTED TEXT** 21 WORDS **83%** **MATCHING TEXT** 21 WORDS

patients had serum albumin >3.5 gm/dL and 10 patients had serum albumin 3.5-4.5 gm/dL. In the

patients had serum albumin less than 3.5 gm/dl and 122 (81.3%) patients had serum albumin ≥3.5 gm/dl. The

W https://www.ijsurgery.com/index.php/isj/article/viewFile/4122/2741

3/4 **SUBMITTED TEXT** 23 WORDS **65%** **MATCHING TEXT** 23 WORDS

patients had serum albumin >3.5 gm/dL, 51 patients had serum albumin 3.5-4.5 gm/dL, 15 patients had serum albumin 4.5-5.5 gm/dL and 1

patients had serum albumin less than 3.5 gm/dl and 122 (81.3%) patients had serum albumin ≥3.5 gm/dl. The mean serum albumin was found to be 3.7 gm/dl±0.32 and

W https://www.ijsurgery.com/index.php/isj/article/viewFile/4122/2741

4/4 **SUBMITTED TEXT** 21 WORDS **73%** **MATCHING TEXT** 21 WORDS

patients had serum albumin >3.5 gm/dL and 1 patient had serum albumin 3.5-4.5 gm/dL. In the

patients had serum albumin less than 3.5 gm/dl and 122 (81.3%) patients had serum albumin ≥3.5 gm/dl. The

W https://www.ijsurgery.com/index.php/isj/article/viewFile/4122/2741

CERTIFICATE BY THE STATISTICIAN

Dr. Debarshi Jana (PhD)
Consultant Statistician
Young Scientist,
Department of Science & Technology,
IPGMER and SSKM Hospital,
Kolkata - 700020.

Contact No- 9836002686
Email.ID:debarsheejana@gmail.com

TO WHOM IT MAY CONCERN

This is to certify that the statistical analysis component of the dissertation titled **'Pre-Operative Serum Albumin as A Predictor of Post-Operative Morbidity and Mortality in Major General Surgeries'** done by Dr. Abhinav Mone, 3rd year post graduate trainee in the Department of General Surgery, Santosh Medical College and Hospital, Ghaziabad, has been conducted under my direct guidance and supervision.

All the statistical calculations of this study are authentic and correct to the best of my knowledge.

Wish him all the success in his life.

Date: 10/11/2022

Debarshi Jana

Dr. Debarshi Jana

Dr. Debarshi Jana
PhD
Young Scientist (DST)
IPGMER and SSKM Hospital
Kolkata

Residence: 14/11, IC, Phase-I, Thakurpukur Hsg Soc, M. G. Road, Kolkata-700104

MASTER CHART

S. No.	IP No.	Name	Age	Gender	Diagnosis	Comorbidity Yes(y)/ No(n)	Malignant(m)/ Non-Malignant(nm)	BMI (Kg/m2)	Hemoglobin (gm%)	Serum Albumin (gm/dL)	Procedure	Complications
1	17154899	abhishek	22	m	acute appendicitis	n	nm	25.7	11.2	4.6	open appendectomy	no
2	17151168	ajay kumar	46	m	fournier's gangrene	y	nm	27.5	12.3	3	debridement	yes
3	13009087	akash sharma	33	m	b/l inguinal hernia with b/l varicocele	n	nm	27.7	11.3	3.8	hernioplasty	no
4	13010595	alka sangal	54	f	WD with SSI	y	nm	32	14.2	2.5	secondary suturing	yes
5	28483	amit kumar	35	m	fournier's gangrene	y	nm	30.5	12	2.8	debridement	yes
6	18871	amit kumar	18	m	acute appendicitis	n	nm	23.5	10.1	4.7	open appendectomy	no
7	30219	asha	45	f	cholelithiasis	n	nm	31.3	10.5	4.2	laparoscopic cholecystectomy	no
8	31915	asha	38	f	cholelithiasis	n	nm	37.7	12	4.24	laparoscopic cholecystectomy	no
9	26298	ashok kumar	52	m	diabetic foot	y	nm	29.1	12	2.5	debridement	yes
10	26407	atar singh	69	m	hydrocele	n	nm	25.4	9.3	5.2	eversion of sac	no
11	31773	badrinath	79	m	recurrent inguinal hernia	n	nm	26.9	9.4	3.6	hernioplasty	no
12	25662	mohan lal	43	m	cholelithiasis	n	nm	32	10.2	4.39	laparoscopic cholecystectomy	no
13	17101753	bhajan lal	49	m	non functional rt kidney	n	nm	18.4	12.3	2.7	nephrectomy	yes
14	30043	bharat singh	75	m	nacrotising facitis	y	nm	30.2	14.3	2.81	debridement	yes
15	25027	seema garg	36	f	supraumbilical hernia	n	nm	36.4	12.3	5.4	hernioplasty	no
16	25070	ajay kumar	45	m	fournier's gangrene	y	nm	31.1	15.3	2.2	debridement	yes
17	20125	jagmohan	48	m	cholelithiasis	n	nm	33.2	10.2	4.4	laparoscopic cholecystectomy	no
18	20102	mohan lal	43	m	cholelithiasis	n	nm	32	14.3	5.3	laparoscopic cholecystectomy	no
19	17172109	rajshree	40	f	cholelithiasis	y	nm	29.1	12.3	3.31	laparoscopic cholecystectomy	no
20	17121710	ramu pandey	30	m	hydrocele	n	nm	22.1	12.2	5.3	eversion of sac	no
21	17172168	deepak	43	m	cholelithiasis	n	nm	30.2	11	4.8	laparoscopic cholecystectomy	no
22	1712105	ranjo	38	f	cholelithiasis	n	nm	32	10.2	6.2	laparoscopic cholecystectomy	no
23	17175126	suneel	54	m	cholelithiasis	y	nm	33.9	12.2	4.3	laparoscopic cholecystectomy	no
24	17148993	shobha	48	f	infected sebaceous cyst rt breast	y	nm	33.6	10.3	5.1	excision	no
25	17157321	pinki malinga	36	f	cholelithiasis	n	nm	32.7	9	3.2	laparoscopic cholecystectomy	no
26	17166481	pooja raghav	25	f	cholelithiasis	n	nm	30.3	9.3	3.4	laparoscopic cholecystectomy	no
27	17177552	praveen	45	m	G4 hemorrhoids	n	nm	24.3	12.3	3.8	conventional hemorrhoidectomy	no
28	17179742	sudha verma	35	f	cholelithiasis	n	nm	33.9	8.5	3.1	open cholecystectomy	no
29	17182271	balbir singh	61	m	cholelithiasis	y	nm	33.1	12.3	5.3	laparoscopic cholecystectomy	no
30	17179842	nasir ali	45	m	sigmoid volvulus	n	nm	18.4	13.2	2.8	EL with RA	yes
31	17184624	bhoolan	66	m	hydrocele	n	nm	27.8	11.5	4.3	eversion of sac	no
32	17183439	anuradha	22	f	cholelithiasis	n	nm	25.9	12.7	4.3	laparoscopic cholecystectomy	no
33	17132128	saddam	18	m	perforation peritonitis	n	nm	21.3	11.9	2.1	EL with grahms patch repair	yes
34	17192685	saheb din	42	m	perforation peritonitis	n	nm	25.7	10.5	2.1	EL with grahms patch repair	yes
35	17191246	ved chand	42	m	hydrocele	n	nm	31.9	14	5.2	eversion of sac	no
36	17192887	sitara devi	32	f	cholelithiasis	n	nm	34.7	12.3	3	open cholecystectomy	yes
37	17223734	shishpal	65	m	mirizzi syndrome	y	nm	17.9	12.4	2.6	open cholecystectomy	yes
38	17225064	shiv kumar	29	m	cholelithiasis	n	nm	28.8	11.8	2.4	open cholecystectomy	yes
39	17225060	dev pratap	18	m	cholelithiasis	n	nm	22.1	10.5	3.8	laparoscopic cholecystectomy	no
40	17217141	shabnam	47	f	fistula in ano	n	nm	46.6	12.6	2.3	LIFT	yes
41	17213303	simmi	60	f	cholelithiasis	n	nm	25.5	10.5	2.8	laparoscopic cholecystectomy	yes
42	17224391	ramesh devi	52	f	cholelithiasis	n	nm	32.7	11.5	2.8	open cholecystectomy	yes
43	17222396	soni	32	f	ventral hernia	n	nm	43.6	12.7	2.4	hernioplasty	yes
44	17224597	neeraj	30	f	cholelithiasis	n	nm	26.6	11.9	2.6	open cholecystectomy	yes
45	17224531	sonu	30	m	perforation peritonitis	n	nm	25.5	10.5	2.2	EL with grahms patch repair	yes
46	17132259	bhoori	47	f	breast abscess	y	nm	33.2	14	2.97	I&D	yes
47	17180499	bhooshan	59	m	inguinal hernia	n	nm	29.7	12.3	4.19	hernioplasty	no
48	17158867	bijendri	32	f	cholelithiasis	n	nm	32.9	12.4	3.28	laparoscopic cholecystectomy	no
49	17166191	chandan	30	m	inguinal hernia	n	nm	20.2	11.8	4.5	hernioplasty	no
50	17202205	lal singh	70	m	hepatic abscess	y	nm	30.3	10.5	3.01	EL with drainage	yes
51	17166336	dayanand	58	m	cellulitis	y	nm	31.2	12.6	3	faciotomy	yes
52	13010801	deepak	27	m	fistula in ano	n	nm	34.7	10.5	4.2	LIFT	no
53	17157242	deepak	30	m	cholelithiasis	n	nm	27.8	11.5	4.16	laparoscopic cholecystectomy	no
54	17143283	deepak	50	m	scrotal abscess	y	nm	28.4	12.7	3.12	I&D	yes
55	17144617	dev karan	70	m	fournier's gangrene	y	nm	27.5	11.9	3	debridement	yes
56	17158116	dharam	19	m	gyanacomastia	n	nm	25.9	10.5	4.02	excision	no
57	17087383	dheeraj	37	m	ischeorectal abscess	n	nm	29.7	14	3.17	I&D	no
58	17112249	dhirwati	50	f	umbilical hernia	n	nm	34.4	12.3	4.2	hernioplasty	no
59	17203920	dinesh	65	m	acute appendicitis	n	nm	15.9	12.4	2.5	open appendectomy	yes
60	17203415	firoz	30	f	cholelithiasis	n	nm	26	11.8	4.2	laparoscopic cholecystectomy	no
61	17100603	niyaz	54	m	diabetic foot	y	nm	32	10.5	3.4	debridement	yes
62	17109907	sunil	40	m	CA urinary bladder	n	m	18	12.6	3.7	resection	no
63	17101753	bhajan lal	49	m	inguinal hernia	n	nm	22.3	10.5	3.4	hernioplasty	no
64	17102053	jay prakash	54	m	CA penis	n	m	21.2	12.2	3.6	penectomy	no
65	17103323	rajendra	40	m	fistula in ano	n	nm	32	11	3.4	LIFT	no
66	17103255	shiv kumar	54	m	cellulitis rt leg	n	nm	33	10.2	3.2	faciotomy	yes
67	17113150	aarti	26	f	LSCS	n	nm	32	12.2	3.2	secondary suturing	yes
68	17113096	simran	37	f	TAH	n	m	33	10.3	3.1	secondary suturing	yes
69	17113450	zabir singh	60	m	inguinal hernia	n	nm	24.5	9	3.6	hernioplasty	no
70	17113472	kuwar pal	65	m	diabetic foot	y	nm	34	9.3	3.5	debridement	no
71	17113124	krunal	35	m	cholelithiasis	n	nm	35	12.3	3.6	laparoscopic cholecystectomy	no
72	17112430	rajesh	45	f	TAH	n	m	36	8.5	3.9	secondary suturing	no
73	17113339	bala	40	f	TAH	n	m	32	12.3	2.8	secondary suturing	yes
74	17110214	ashutosh	37	m	fissure in ano	n	nm	34	13.2	3.4	LIS	no

75	17109708	roshan lal	70	m	grade 3 hemorrhoids	n	nm	33	11.5	3.6	MIPH	no
76	17114197	rajesh	41	m	grade 3 hemorrhoids	n	nm	28	12.7	3.2	conventional hemorrhoidectomy	no
77	17112267	neeraj	32	m	grade 3 hemorrhoids	n	nm	22	11.9	3.4	MIPH	no
78	17111539	suresh pal	49	m	grade 3 hemorrhoids	n	nm	20	10.5	3.9	MIPH	no
79	17102396	pradeep	30	m	grade 3 hemorrhoids	n	nm	18	12.2	3.7	MIPH	no
80	17105141	tamilarasan	46	m	cholelithiasis	n	nm	32	11	3.8	laparoscopic cholecystectomy	no
81	17105188	surendra	45	m	cholelithiasis	n	nm	33	10.2	3.4	laparoscopic cholecystectomy	no
82	17105142	sharma	55	m	cholelithiasis	n	nm	36	12.2	3.3	laparoscopic cholecystectomy	no
83	17105166	harshit	39	m	CA GB	y	m	32	10.3	3.3	open cholecystectomy	no
84	17105144	harshit	36	m	incisional hernia	n	nm	28	9	3.3	laparoscopic hernioplasty	no
85	17105096	kuljeet	34	f	LSCS	y	nm	33	9.3	2.8	secondary suturing	yes
86	17105161	singh	36	m	incisional hernia	n	nm	26	12.3	3.6	laparoscopic hernioplasty	no
87	17105129	manisha	41	f	TAH	y	m	32	8.5	2.8	secondary suturing	yes
88	17105099	bimlesh	40	f	TAH	n	m	28	12.3	3.3	secondary suturing	yes
89	17105160	kumar	39	m	grade 3 hemorrhoids	n	nm	23	13.2	3.3	conventional hemorrhoidectomy	no
90	17107821	mustafa	34	f	LSCS	n	nm	22	11.5	3.6	secondary suturing	yes
91	17105199	rahul	45	m	gyanacomastia	n	m	33	12.7	3.4	excision	yes
92	17105183	subhash	45	m	diabetic foot	y	nm	32	11.9	2.4	debridement	yes
93	17107818	saroja	34	f	post MRM	y	m	33	10.5	2.6	secondary suturing	yes
94	17107835	munni	37	f	breast abscess	n	nm	23	12.2	3.4	I&D	yes
95	17105148	krunal	36	m	post MRM	y	m	20	11	2.6	secondary suturing	yes
96	17107297	om narayan	68	m	grade 3 hemorrhoids	n	nm	18	10.2	3.6	conventional hemorrhoidectomy	no
97	17107833	pallavi	48	f	TAH	n	m	33	12.2	3.3	secondary suturing	yes
98	17105203	vijay	43	m	BPH	n	m	23	10.3	3.6	open prostatectomy	no
99	17107826	manju	23	f	LSCS	n	nm	26	9	3	secondary suturing	yes
100	17105177	shankar	45	m	nacrotising facitis	n	nm	25	9.3	2.2	debridement	yes
101	17105174	sakir m	46	m	incisional hernia	n	nm	22	12.3	3.3	hernioplasty	no
102	17105179	chand	55	m	wound dehiscence	y	nm	36	8.5	3	secondary suturing	yes
103	17099948	amzad	40	m	diabetic foot	y	nm	33	12.3	3.1	debridement	yes
104	17105196	saroja	33	f	LSCS	n	nm	17	13.2	2.6	secondary suturing	yes
105	17112049	mohit	43	m	grade 3 hemorrhoids	n	nm	23	11.5	3.3	conventional hemorrhoidectomy	no
106	17106377	pushpa	35	f	cholelithiasis	n	nm	24	12.7	4.29	laparoscopic cholecystectomy	no
107	17204289	radha	38	f	cholelithiasis	n	nm	32.9	11.9	4.1	laparoscopic cholecystectomy	no
108	17193397	radhika	40	f	post LSCS wound dehiscence	n	nm	27.2	10.5	2.8	secondary suturing	yes
109	13011427	rajkumar	47	m	fournier's gangrene	y	nm	31.2	12.2	2.4	debridement	yes
110	17152107	rajshree	25	f	cholelithiasis	n	nm	24.4	11	3.31	laparoscopic cholecystectomy	no
111	17123781	raju	47	m	ruptured liver abscess with peritonitis	y	nm	21	10.2	3.35	EL with peritoneal levage with drainage of liver abscess	yes
112	17151476	ramwati	68	f	SAIO	n	nm	32.8	12.2	3.42	EL with RA	no
113	17157540	ranjo devi	35	f	cholelithiasis	n	nm	28.4	10.3	4.18	laparoscopic cholecystectomy	no
114	17182276	ravita	35	f	hepatitis with AIO with hypoglycemic encephalopathy with AKI	y	nm	29.6	9	2.3	EL with RA	yes
115	17154698	reena	33	f	cholelithiasis	n	nm	27.9	9.3	4.16	laparoscopic cholecystectomy	no
116	17117528	rohit	18	m	acute appendicitis	n	nm	27.1	12.3	3.38	laparoscopic appendectomy	no
117	17206743	sagar	18	m	perforation peritonitis	n	nm	17.8	8.5	3.21	EL with RA	yes
118	17147508	sandeep	51	m	carbuncle	n	nm	21.4	12.3	4.2	excision	no
119	17106584	sandeep	32	m	CA GB	n	m	26.1	13.2	3.7	open cholecystectomy	no
120	17204047	sapna	31	f	cholelithiasis	n	nm	29.1	11.5	4.6	open cholecystectomy	no
121	17157198	saroja	48	f	cholelithiasis	n	nm	26.2	12.7	3.48	laparoscopic cholecystectomy	no
122	17157951	satbir	55	m	inguinal hernia	n	nm	19.6	11.9	4.2	hernioplasty	no
123	17152012	satish	54	m	CA colon	n	m	18.9	10.5	2.1	hemicolectomy	yes
124	17108534	saurabh	22	m	grade 3 hemorrhoids	n	nm	18.9	12.2	4.6	MIPH	no
125	17137684	seema garg	36	f	supraumbilical hernia	n	nm	26.1	11	3.18	hernioplasty	no
126	17092226	seema tomar	39	f	ileal stricture	n	nm	27.2	10.2	3.4	EL with RA	no
127	17199907	shimanshi	44	f	cholelithiasis	n	nm	26.2	12.2	3.49	laparoscopic cholecystectomy	no
128	17154267	shikha	18	f	phylloides tumour	n	m	26.1	10.3	4.21	excision	no
129	17158993	shobha	40	f	infected sebaceous cyst rt breast	n	nm	28.2	9	3.8	debridement	no
130	17129770	surbhi	25	f	cholelithiasis	n	nm	29.1	9.3	4.35	laparoscopic cholecystectomy	no
131	17188436	soni	40	f	ventral hernia	n	nm	26.1	12.3	3.5	hernioplasty	no
132	17220374	bhageshwari	51	f	cholelithiasis	n	nm	31.2	8.5	3.8	laparoscopic cholecystectomy	no
133	17223075	rohit	40	m	inguinal hernia	n	nm	29.1	12.3	2.8	hernioplasty	yes
134	17215993	lucky	20	m	pilonidal sinus	n	nm	37.2	13.2	3.1	excision with rhomboid flap repair	yes
135	17222950	rajendra	30	m	varicose vein	n	nm	27.2	11.5	2.6	venous stripping	yes
136	17221584	abhay	78	m	wound dehiscence	y	nm	27.2	12.7	2.5	secondary suturing	yes
137	17222957	janki	55	f	cholelithiasis	n	nm	38.2	11.9	3.6	laparoscopic cholecystectomy	no
138	17218079	rahul	18	m	grade 2 varicocele	n	nm	19.8	10.5	3.6	varicocelectomy	no
139	17099361	gorakh	65	m	cholelithiasis	n	nm	23.4	12.2	3.21	laparoscopic cholecystectomy	no
140	17147443	habib	60	m	perforation peritonitis	n	nm	22	11	2.6	EL with RA	yes
140	17140721	iqlash	42	m	CA tongue	n	m	22.1	10.2	3.9	commando operation	no
142	17009969	imran	34	m	cholelithiasis	n	nm	29.4	12.2	3.4	laparoscopic cholecystectomy	no
143	17202990	iirfan	48	m	cholelithiasis	n	nm	29.2	10.3	4.2	laparoscopic cholecystectomy	no
144	17110015	jagmohan	46	m	CA GB	n	m	26.1	9	2	open cholecystectomy	yes
145	17186122	jagresh	52	f	cholelithiasis	n	nm	26.6	9.3	3.8	laparoscopic cholecystectomy	no
146	17159026	jaiwati	54	f	CA GB	n	m	21.2	12.3	3.19	open cholecystectomy	yes
147	17188196	janki	39	f	supraumbilical hernia	n	nm	27	8.5	4.05	hernioplasty	no
178	17188098	juhi	28	f	choledocholithiasis	n	nm	31.4	12.3	3.8	choledochotomy	no
149	17166502	kamlesh	45	f	perforation peritonitis	n	nm	27.7	13.2	3.47	EL with grahms patch repair	no
150	17187993	krishna devi	47	f	PIVD	n	nm	31.1	11.5	3.75	interlaminar disectomy	no

151	17204269	kuwar pal	63	m	CA tongue	n	m	32.8	12.7	2.57	commando operation	yes
152	17203826	man singh	62	m	CA thyroid	y	m	17.6	11.9	2.5	total thyroidectomy	yes
153	17203551	madhuri	35	f	cholelithiasis	n	nm	30.5	10.5	3.4	laparoscopic cholecystectomy	yes
154	17102803	mahadevi	60	f	CA breast	n	m	28	12.2	3.5	MRM	yes
155	17199963	mahendra pal	45	m	fissure in ano	n	nm	30	11	3.63	LIFT	no
156	17203986	manoj	25	m	obstructive uropathy	n	nm	27	10.2	2.8	PCNL	yes
157	17177575	manoj	40	m	cholelithiasis	n	nm	24.8	12.2	3.8	laparoscopic cholecystectomy	no
158	17203937	meena	38	f	cholelithiasis	n	nm	25.6	10.3	4.88	laparoscopic cholecystectomy	no
159	17174842	nasir ali	45	m	sigmoid volvulus	n	nm	22.3	9	4.8	EL with RA	no
160	17203904	neeraj	32	m	cholelithiasis	n	nm	29	9.3	3.1	laparoscopic cholecystectomy	yes
161	17118778	neeraj	35	m	acute appendicitis	n	nm	27.2	12.3	2.56	EL with peritoneal levage with appendectomy	yes
162	17203925	naman	32	m	marjolins ulcer	n	m	26	8.5	3	WLE	yes
163	17169501	nitish	23	m	CA GB	n	m	28	12.3	3.35	open cholecystectomy	no
164	17100602	niyaz	45	m	diabetic foot	y	nm	28.2	13.2	3.32	Amputation	yes
165	17099138	pallavi	18	f	morel lavalli	n	nm	29.9	11.5	3.1	I&D	yes
166	17164420	pankaj	29	m	cholelithiasis	n	nm	26	12.7	3.8	laparoscopic cholecystectomy	no
167	17157321	pinki malinga	36	f	cholelithiasis	n	nm	23	11.9	3.81	laparoscopic cholecystectomy	no
168	17201546	pooja raghav	23	f	wound dehiscence	n	nm	19.9	10.5	2.7	secondary suturing	yes
169	17166481	pooja	25	f	cholelithiasis	n	nm	19.8	12.2	3.68	laparoscopic cholecystectomy	no
170	17148008	poonam	30	f	ileocecal TB	n	nm	22.7	11	3.62	EL with RA	no
171	17133770	poonam	45	f	CA colon	n	m	25.6	10.2	2.6	hemicolectomy	yes
172	17130088	prashant	28	m	acute appendicitis	n	nm	24.8	12.2	3.4	laparoscopic appendectomy	no
173	17177552	praveen	45	m	grade 3 hemorrhoids	n	nm	25.3	10.3	3.95	MIPH	no
174	17158315	prem	48	m	cholelithiasis	n	nm	32	9	3.5	laparoscopic cholecystectomy	no
175	17170912	priyanka	36	f	marjolins ulcer	n	m	32.7	9.3	3.7	WLE	no
176	17225199	babli	38	f	cholelithiasis	n	nm	32.4	12.3	3.4	laparoscopic cholecystectomy	no
177	17154804	priyanka	18	f	AIO	n	nm	17.8	8.5	3.4	EL with RA	no
178	17151103	shreyash	70	m	BPH	y	m	18.9	12.3	2.9	open prostatectomy	yes
179	13009037	shraddha	60	f	CA breast	y	m	32	13.2	2.8	MRM	yes
180	13010582	bikki	18	m	acute appendicitis	n	nm	18.3	11.5	3.4	laparoscopic appendectomy	no
181	18875210	ilka	34	f	fibroadenoma	n	m	19.9	12.7	3.39	excision	no
182	31819369	dharam	80	m	CA tongue	n	m	20.8	11.9	3	commando operation	yes
183	17125456	wasim	63	m	cholelithiasis	n	nm	33.5	10.5	3.4	laparoscopic cholecystectomy	no
184	17102365	aayush	27	m	CA penis	n	m	20.9	11.2	3.4	penectomy	no
185	17120359	sayra	25	f	fibroadenoma	n	m	33.5	12.3	3.6	excision	no
186	17123025	animesh	45	m	ventral hernia	n	nm	33.4	11.3	3.1	hernioplasty	yes
187	17156428	sujeet	55	m	CA colon	n	m	17.8	14.2	2.6	hemicolectomy	yes
188	17125698	amit kumar	56	m	CA colon	n	m	18.9	12	2.5	hemicolectomy	yes
189	17125698	sumit	78	m	CA tongue	n	m	19.3	10.1	2.8	commando operation	yes
190	17159852	chinmay	80	m	CA penis	n	m	17.9	10.5	2.9	penectomy	yes
191	17156565	prateek	66	m	CA prostate	y	m	18.5	12	3	prostatectomy	yes
192	17156464	saurabh	45	m	gyanacomastia	n	m	34.6	12	3	mastectomy	no
193	17164564	gaurav	63	m	CA buccal mucosa	y	m	19.9	9.3	2.7	commando operation	yes
194	17164654	ruchika	65	f	CA breast	n	m	34.2	9.4	2.6	MRM	yes
195	17131246	ruchiira	54	f	CA ovary	y	m	33.2	10.2	2.9	hysterectomy	yes
196	17164595	jahnavi	52	f	CA breast	n	m	26.4	12.3	2.8	MRM	yes
197	17165469	sonalika	78	f	CA breast	y	m	17.9	14.3	2.5	MRM	yes
198	17164654	banti	79	m	CA colon	y	m	17.3	12.3	2.8	hemicolectomy	yes
199	17146465	bulbul	45	f	fibroid uterus	n	m	32.3	15.3	3.4	hysterectomy	no
200	17146545	nileshwar	55	m	CA tongue	n	m	22	10.2	3.6	commando operation	no